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"Scire est nescire, nisi id me
Scire alius sciret."

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ON THE COURSE OF PERNICIOUS ANÆMIA IN OLDER PERSONS.

BY

J. MICHELL CLARKE, M.A., M.D. Cantab., LL.D. Bristol, F.R.C.P.,

Professor of Medicine, University of Bristol;
Physician, Bristol General Hospital.

IN a disease like pernicious anæmia, which after all is not very common, it is difficult for one observer to get a sufficient number of examples from which to draw any general conclusions from his personal observations. Moreover, in hospital work, the opportunity to follow the course of a chronic disease for any considerable time is rare, and especially so in pernicious anæmia, in which the course of the illness is characterised by marked exacerbations, during which the patient comes into hospital and is lost to sight during the intervals. To get a full knowledge of pernicious anæmia frequent observations on the blood are necessary throughout the course of the illness, including the remissions. The majority of cases of pernicious anæmia occur between the ages of 35 to 55 years, but for some time I had noticed in a few cases in elderly people that the disease appeared to follow

a somewhat different course with especial features in the blood-changes, yet as they were not observed during long, continuous periods, it was possible that the variations observed might be only individual and accidental. Lately, however, I have had the opportunity of closely following the course of two cases in elderly men for about twelve months in each case, and in a third for six months.

In this paper I propose to deal with some features of pernicious anæmia where it occurs in elderly or old persons, of which I have eight cases available, and to compare them with cases occurring between the ages of 30 and 50, of which I have taken for comparison fifteen recent cases. The observations rest on a very large number of blood-counts, made or verified personally, and of which only the averages can be given in this paper. The hæmoglobin was estimated by Gowers's method or by Haldane's modification. Leishman's stain was used for films, supplemented by Jenner's stain when it seemed desirable. I do not propose to discuss at length what is and what is not true pernicious anæmia, other than to say that the evidence is in favour of its being a disease of the marrow brought about by the action of toxins varying in nature, the features of the disease being more or less constant and easily recognisable, due not to actual identity of the exciting cause in each case, but to the fact that the cause acts always primarily upon one and the same highly-specialised tissue, the marrow, which responds in a constant way. The most important changes which give the disease its characteristic clinical features are of course found in the blood, and of these the high colour index and the presence of macrocytes in large numbers seem to me the most important single changes, because the most constant, and because they are not regularly found in other forms of anæmia. To get their full significance, however, one or two blood-counts in a particular case are not sufficient, as occasionally the colour-index, especially in chronic cases, falls below 1. I am also of opinion that there is still something to be learnt as to the nature of pernicious anæmia from the variations in the number and variety of the leucocytes, and I have paid especial attention to this point.

In the type of the disease in elderly persons, which conforms to what has been described as the chronic type of pernicious anæmia, but which, though during the main part of its course it is of moderate severity, is not extremely protracted in duration, the etiology is as obscure and the onset as insidious as in other forms. In such persons most of the teeth have often been lost, and the remaining ones, especially the lower incisors, are loose with receding gums. In one case only the patient had had definite pus-formation, but this had been cured by extraction of the teeth and vaccines twelve months before the appearance of the distinctive anæmia. In three others without evidence of pus-formation cultures taken deeply from the root of the loosened teeth, after carefully cleansing the gums and superficial parts, only gave the presence of micro-organisms which Dr. Scott Williamson, pathologist to the hospital, considered were not pathogenic. Removal of the teeth had no effect on the course of the anæmia. In the other cases the teeth and gums were healthy, and this corresponds to my experience of pernicious anæmia generally—that the condition of the teeth is sometimes good, sometimes bad, but too variable to be of absolute significance in the origin of the disease. In several of the cases the patients were in easy circumstances, leading healthy lives under good hygienic conditions.

The duration in these cases is about twelve months, and the anæmia is definitiely established by the time the patient seeks medical advice. At first there is not the great loss of strength which occurs early and is so marked a symptom in the more acute forms of pernicious anæmia, and for a long time the patient is able to get about and even to do a moderate amount of work without great fatigue and in fair comfort, except for some shortness of breath and palpitation. After a few months, however, loss of strength becomes marked, and then steadily increases until the fatal termination, which occurred in six of the eight cases ; one I could not trace.

Although there are intervals in which the condition, both general and of the blood, improves, there are not the marked remissions which are so characteristic a feature of the more

common type of pernicious anæmia, and on the whole, with occasional intervals of improvement, the course of the disease is gradually but steadily progressive. Gastro-intestinal symptoms, which are so frequent in all forms of pernicious anæmia, so difficult to treat, and whose frequency and severity are of such great importance to the progress of the case, are especially prominent in these cases, in the form of attacks of vomiting and diarrhœa, and especially of nausea, dryness of the mouth, and profound anorexia. This dryness of the mouth and anorexia are particularly difficult of relief. Probably, chiefly from these causes, there is a steady loss of flesh in the latter months of the illness, which becomes rapid towards the end. Beyond some tingling and numbness in feet and legs, signs of spinal affection were present in only one of these cases.

The patients show early and markedly the characteristic lemon-coloured tint of skin, and often a certain amount of scattered pigmentation. Hemorrhages do not occur except in the form of retinal hemorrhages, which were present in my cases. The spleen was either not enlarged or very slightly so. In ordinary and more acute cases of pernicious anæmia I regard the presence of a well-marked urobilin band in the spectroscopic examination of the urine as a valuable confirmatory aid in the diagnosis, but in these more chronic cases it was only occasionally found. Towards the end of the illness the blood-pressure is apt to fall very low, possibly indicating exhaustion of the supra-renals. As to the condition of the blood in these eight cases the changes are of moderate severity, corresponding to those of the more chronic type of pernicious anæmia, and is given in the third line (*c*) of the Table II, p. 105, where it is seen that the red cells average about two millions, hæmoglobin 40 per cent., total leucocytes 5720 per c.m., of which 35 per cent. are small lymphocytes and 9.5 large mononuclears. In line (*e*) two cases are given separately, as blood-counts were continuously taken at intervals of a fortnight during nine and twelve months respectively, and in (*d*) the six other cases, which were only seen for shorter periods. The condition of the blood in the prelethal stage (one week or so before death) is given separately for all

these cases in line (*g*). The difference between (*c*), (*d*) and (*e*) illustrates what was said in the beginning as to the necessity of more continuous observations on the blood in cases of anæmia than we are able to secure in hospital practice, where the patients come in for short periods when they are at their worst, and also in private practice, where ordinarily the blood is only examined occasionally at arbitrary intervals. It follows from this that (*d*), taken from patients discontinuously observed, gives too unfavourable a representation of the blood-state during the whole course of the illness, and that the average of red cells, hæmoglobin and leucocytes is seen to be higher when the cases continuously observed are taken into account.

The accompanying chart, Table I, brings out this point, and shows the course of the disease in the two cases continuously followed, an average being taken for each month. The chief points are that the red corpuscles and hæmoglobin follow corresponding curves and remain at a low, though not a very low, level during the whole course of the illness, and without any great remissions or variations until near the end, when they both show a rapid and steady fall until death occurred.

The curve of the small lymphocytes is also noteworthy, as it fairly closely follows that of the red cells and hæmoglobin, and temporary improvements in the case coincided with an increase in the percentage of small lymphocytes. I have noticed this also in myeloid leukæmia, improvement under different forms of treatment coinciding with an increase of these cells. Of course, it is to be borne in mind that in nearly all cases of pernicious anæmia there is a leucopenia, together with a relative increase in the lymphocytes and mononuclears, especially in the more chronic cases, and allowance must be made for this.

There are extraordinary variations in the total leucocytes for which I could not account, and which could not be accounted for by any variation in the clinical symptoms, nor do they correspond with the small variations in amounts of red cells and hæmoglobin. There is a marked rise of leucocytes in each case shortly before death. The polymorphonuclears show a remote correspondence with the variations in total number of leucocytes.

TABLE I.

Case A.	Red Cells.	Hæmo-globin.	Colour Index.	Total Leucocytes.	Polymorpho-nuclears.	Small lymphocytes.	Large lymphocytes and mononuclears.	Eosinophils.
	Per c.m.	%	%	Per c.m.	%	%	%	%
1911. Sept.	1,700,000	50	1.4	8,000	53	34	18	4
Oct.	2,100,000	50	1.2	7,600	72	16	10	2
Nov.	3,700,000	60	.8	7,500	62	22	14	1
Dec.	3,100,000	60	1	10,000	59	29	7.4	.6
1912. Jan.	2,500,000	54	1.+	10,000	69	23	7	.3
Feb.	3,800,000	70	1.+	12,000	62	25	12	.2
March 5th	2,400,000	45	.9	4,500	49	39	11	.2
March 20th	1,100,000	28	1.3	10,700	69	27	3	0
Case B.								
1911. Nov.	2,800,000	62	1.1	8,400	42.5	42	12	1.5
Dec.	3,000,000	70	1.1	7,000	39.5	45.5	15	1
1912. Jan.	3,200,000	70	1	6,000	42	49	7.5	1
Feb.	3,700,000	70	.9	6,000	51	35.5	10.5	1.5
March.. ..	3,100,000	68	1.1	11,500	39	38	19	3
April	2,200,000	60	1.3	5,000	36	44.5	17	1.5
May	2,900,000	60	1	4,300	37	43	16	2
June	2,800,000	62	1.1	9,000	64	20	14.5	2
July	3,400,000	55	.8	9,200	—	—	—	—
August ..	1,300,000	28	1.1	6,500	65	18	12	2
Sept.	860,000	25	1.4	3,400	54	38	7.5	0
Oct. 5th ..	912,000	25	1.4	10,000	52	33	12	0

Possibly these variations indicate an attempt on the part of the organism to react to the entrance of fresh doses of toxin.

Both these patients were treated at intervals with 10 to 20 c.c. doses of polyvalent antistreptococcic serum per rectum, but I could not trace out any effect from these injections on either the number or the percentage variety of the leucocytes.

To return to the eight cases taken together. The large mononuclears, though slightly increased in numbers, showed no striking variations. In these cases, however, these cells were generally of great size, and had large and very irregular nuclei, the nuclei having many lobes and often several vacuoles in them, the whole giving the impression that the cells were in a very active state. These are amœboid cells, and possibly show an attempted reaction on the part of the organism to infections or poisons with which the polymorphonuclears are unable to deal.

An important point about these cases in elderly persons is that both megaloblasts and normoblasts are either absent throughout the course of the disease until just before the end or present in very scanty numbers. This is true of chronic cases of pernicious anæmia generally, according to Gulland and Goodall, and is certainly a marked feature of the cases under consideration. This is one of the chief differences from pernicious anæmia in younger patients, or in the more ordinary acute or subacute forms, and might lead to an error of diagnosis if the presence of megaloblasts be taken as the criterion of the disease. Again, from the point of view of those who look upon pernicious anæmia as essentially a megaloblastic anæmia, this point might be considered sufficient to exclude these cases from the category. However, the fact that in the late stages there is an irruption of these cells into the blood, often in large numbers, thus bringing the condition of the peripheral blood into conformity with the general type, is a sufficient answer to this objection, for there could be no difference of opinion as to the films taken in the final stages presenting the blood-picture of megaloblastic pernicious anæmia. Macrocytes are numerous throughout, and my experience confirms the statement of Buckmaster, that of the abnormal changes in the blood

this is the most constant in all forms of pernicious anæmia ; I would add in association with a high colour-index.

Blood platelets are scanty in these cases. Chromatoplasia and granular degeneration of the red corpuscles, though generally present at intervals and towards the end, are not marked features. A few myelocytes, 1 per cent. or less, are found. In Table II (*c*) is given the condition of the blood in these eight cases, and in (*a*) that of fifteen cases of pernicious anæmia below the age of fifty, several of them of acute type, based on an average of numerous blood-counts taken in all phases of the disease. It is seen that they differ in the following points.

In the former (*c*) the number of red cells is greater, percentage of hæmoglobin slightly higher, not so much in proportion as the number of red-cells, and the colour-index lower, whilst the number of leucocytes is much greater, and the relative proportion of small lymphocytes and mononuclears is also higher. The contrast as regards leucocytes and lymphocytes would be even more marked but that the leucopenia happens in the fifteen cases in (*a*) to be less than the average usually given for ordinary cases of pernicious anæmia.

The contrast between the two types becomes far more striking, except as to one point, if the blood-state of the eight cases in (*c*) is compared with that in (*b*), which represents the average of a number of blood-counts from cases during the severe exacerbations which characterise the course of ordinary acute and subacute pernicious anæmia, and at the same time we remember that such severe exacerbations were absent in the eight older and more chronic cases (*c*). In the exacerbations the red cells and hæmoglobin, as compared with (*c*), are now reduced still further, to about half, the colour-index is correspondingly higher, and the leucopenia very marked. On the other hand, there is one point in whiich the two conditions now approximate, and that is in the proportion of lymphocytes present, for these show a temporary but marked increase during exacerbations of the disease. In both (*a*) and (*b*), especially in the latter, the difference between them and (*c*) in the matter of megaloblasts and normoblasts is noteworthy.

TABLE II.

	Red cells.	Hæmoglobin.	Index.	Total Leucocytes.	Polymorphonuclears.	Small lymphocytes.	Large lymphocytes and mononuclears.	Eosinophils.	Basophils.	Macrocytes.	Megaloblasts.	Normoblasts.	
		%	%		%	%	%	%	%				
(*a*) Blood from average of 15 cases under 50 years of age.	1,575,000	36.8	1.2 —1.3	4,500	67	24	6.7	.9	.4	numerous.			(*a*)
(*b*) The same during severe exacerbations.	964,000	24	1.3	2,400	52.9	42	4	.3	.2	numerous	about 20%	numerous.	(*b*)
(*c*) Blood from average of 8 cases over 55 years of age.	2,120,000	40	1	5,720	53	35	9.5	2.4	.4	,,	few or absent.		(*c*)
(*d*) Blood from average of 6 cases in (*c*) over 55 years of age.	1,754,000	35	1+	5,000	55	34	8	2.7	.5	,,	absent or very few.		(*d*)
(*e*) The same in 2 cases followed for last 12 months of life.	2,680,000	48	.9	7,800	60	29	9.4	1.4	.2	,,	.3%	.2%	(*e*)
	2,800,000	59	1.1	8,300	46	37.5	13.5	1.8	.4	,,	absent.	.4%	
(*f*) The blood in prelethal state (within 48 hours of death) in cases under 50 years of age.	850,000	17	1.8	2,400	54	35	10.2	.2	.2	,,	10-40%	numerous.	(*f*)
(*g*) The same in prelethal states in cases over 55 years of age.	1,000,000	25	1.3	9,700	55	31	12	.3	.4	,,	12%	3.5%	(*g*)

The blood-state in severe exacerbations may be as bad as or worse than that seen in the prelethal stage of pernicious anæmia, *i.e.* within twenty-four to forty-eight hours of death, and it is interesting to note that patients may recover from such exacerbations and afterwards enjoy a long remission of comparatively good health, and that they often die with a condition of blood not so bad as it may have been in a previous exacerbation from which they made a good recovery.

I have had the opportunity of examining the blood in a number of cases of both the acute and chronic (or senile) type within twenty-four to forty-eight hours of death. The condition of the blood may then be taken to represent the ultimate result of the disease on the marrow. However great the differences in the blood between the two forms during the previous course of the disease in the prelethal stage they closely approximate. The table (*f*) (*g*) gives the results in the two types. There is still a difference in severity, but now slighter as regards red cells, hæmoglobin and colour-index; the chief difference is in the total leucocyte count, which rises towards death in the chronic or senile cases; the relative numbers of the varieties of leucocytes closely approximate, the percentage of lymphocytes and mononuclears being high in both; and there is now an irruption of megaloblasts and normoblasts into the blood in the chronic cases. This took place during the last two or three weeks of life in my cases and increased until death. Thus, as stated above, in the final stage of all forms of pernicious anæmia the blood-state is practically identical, and it follows that blood examinations made then would not show the differences found earlier.

Thus in the final stage of the type of case under consideration the blood-changes at last present the full picture of that of pernicious anæmia, with the difference that runs through the course of the case that there is no marked leucopenia and even a slight increase in the number of leucocytes. That this difference and the greater variations in the leucocyte count than occur in acute or subacute cases in younger patients may be due either to a different etiology or to a difference in reaction on the part of

the marrow is obvious. There is no evidence to decide between these alternatives. Whatever the poison at work, it would seem to be combated rather by the lymphocytes and large mononuclears than by the polymorphonuclears.

A great deal can be done by treatment in mitigating the course and severity of the disease, though unfortunately the effects are not lasting. When the patient first comes under observation rest in bed is the first essential point, and next careful regulation of the diet with appropriate remedies for gastro-intestinal disturbances if these are present, as is generally the case. It must be seen that a sufficient amount of easily-digested food is taken, and the diet should include green vegetables and the juices of fruit. It need hardly be said that arsenic is the drug from which most benefit is derived, given not continuously but for definite periods, in gradually-increasing doses. In the type of case under consideration iron, preferably in the form of one of the so-called organic preparations, is certainly useful.

Several of the patients were treated from time to time with serum, and showed a marked improvement under it, in the earlier stages of the illness. This was manifested by an improved general sense of well-being and gain in strength, corresponding with amelioration in the state of the blood. As stated above, however, I could not connect any definite feature of the blood with the use of serum. It was not given in relation to the presence or absence of pyorrhœa. At first I used antistreptococcic polyvalent serum, later horse serum. It was given per rectum in doses of 10 to 15 c.c. daily for one to two weeks, then every other day for two to four weeks.

Two cases were treated by salvarsan ; the one, a man, aged 62, showed a remarkable improvement. A mean of several counts, the blood before salvarsan, gave red cells 1,150,000, hæmoglobin 32 per cent., index 1.4, leucocytes 6,300, megaloblasts and monoblasts 2.4 per cent. Salvarsan was injected on July 6th. After steady improvement and gain of weight the counts gave on July 30th reds 4,200,000, hæmoglobin 65 per cent., index .8, leucocytes 10,700, no nucleated red cells.

Unfortunately I have not been able to trace this patient, so that I cannot say whether he has remained well. In the other case there was little benefit.

METHODS OF DIAGNOSIS IN GASTRIC CANCER.

BY

J. M. FORTESCUE-BRICKDALE, M.A., M.D. Oxon.,

Assistant Physician to the Bristol Royal Infirmary and Clinical Lecturer in the University of Bristol.

THE importance of early diagnosis in cases of cancer of the stomach has given rise to a large amount of clinical and laboratory work, the object of which has been to produce some test or sign so characteristic of the early stages of the disease as to make it possible for a physician to recommend and a surgeon to employ operative treatment at a time when it is likely to be of positive advantage to the patient. I propose to consider the value of the various tests which have been devised, and in order to do so it will be convenient to classify them into three main groups. In the first group I would place those tests which are applied to the contents of the stomach; in the second, those applied to the blood; and in the third, those applied to the urine.

I. TESTS APPLIED TO THE GASTRIC CONTENTS.

These may be subdivided into three smaller groups—

1. Those which show stasis of the stomach contents.

These are three in number, namely the absence of hydrochloric acid,[1] the presence of lactic and other organic acids, and the presence of the so-called Opler-Boas bacilli.

2. In the second group are those tests which show the presence of ulceration, namely the various methods for demonstrating minute quantities of blood in the gastric contents or

fæces [2] and the tests for serum albumen in the stomach washings (Salomon's test). [3]

These tests have been introduced for some years, and their value and limitations are now well recognised. [4] The demontration of stasis or of an ulcerated surface is a valuable additional item in diagnosis, but their presence or absence are of no preponderating or conclusive significance. They may not even be early occurrences in the course of a gastric cancer, so that no special reliance can be placed on their presence or absence as indications for or against operation.

3. The third group consists of a number of tests for alterations in the gastric contents, due to the special influence of carcinomatous growths.

(*a*) *Gluzinski's test.* [5]—When an ulcer heals or becomes carcinomatous the HCl fails and a mucous gastritis supervenes. Gluzinski considers that the former occurrence is so rare as to be negligible. The stomach contents have to be removed three times on the same day and tested for free HCl and total acidity. The published cases do not seem very convincing.

(*b*) A more simple test is that for dissolved albumins. [6] Wolff states that in simple achylia only a small amount of the albuminous matter in a test meal is dissolved, whereas in carcinoma a high percentage of dissolved albumin is found. The reagent used is phosphotungstic acid, prepared according to a formula devised by Wolff, and is added to a series of dilutions of the filtered gastric contents. If much albumen is present high dilutions will, of course, show a cloudy precipitate and vice versa. The only difficulty about the practical value of this test is that, though the findings are clear enough in undoubted cases of cancer, the border-line cases are not so easily decided. Medium degrees of dissolved albumin may or may not indicate cancer. The finding of very small amounts is no doubt very much against the diagnosis.

(*c*) It has been suggested that the reason why there is often little free HCl in the gastric contents in cases of cancer is that the albumens are split up into amino-acids, which combine with more HCl than the original large protein molecule.

However this may be, it seems clear that under certain conditions increased splitting of proteins does occur in cancer of the stomach, and this is the basis of the well-known tryptophane test.[7] This test is apt to be interfered with by the presence of blood or bile in the stomach. Blood in dilution of 1 in 500 may upset the test, and in greater amounts certainly will do so. Regurgitation of trypsin from the duodenum and increased protein-splitting by bacterial action may also vitiate this test. Moreover, it sometimes gives a negative result in early cases, and may give a positive result in other conditions, such as ulcer and simple achylia. If a number of tests are made, however, a series of negatives may be taken as very much against cancer. Schryver and Singer,[8] in the autumn of last year, published some observations showing that a ferment capable of splitting peptone occurred in numerous conditions of severe gastric disease, that it was not characteristic of cancer, and was most commonly associated with dilatation and achylia.

(*d*) The toxicity of the gastric juice to guinea-pigs has also been employed as an early test for gastric cancer. Liveriato[9] injected it subdurally, and found that whereas 1 c.c. from normal stomachs produced no symptoms, 0.1 c.c. from cancer cases was sometimes fatal. Anaphylaxis was produced by the maximum harmless dose of 0.05 c.c., which never occurred with normal gastric juice. The guinea-pigs were prepared by an injection of extract of carcinoma, and then given subdurally a small amount of filtered gastric juice, obtained three-quarters of an hour after an ordinary Ewald test meal. A marked symptom in the anaphylactic shock produced was a fall of temperature. Control experiments with normal gastric juice and with non-sensitised animals were negative. Similar reactions have been obtained by other workers for blood serum, and not only in cancer but in syphilis, tuberculosis and other diseases. The reaction in any case appears to be against the foreign cells and not against the specific cause of cancer, whatever that may be. Embryonic cells, other neoplasms, and apparently normal sera occasionally give anaphylactic reactions in the sensitised guinea-pigs.

(*e*) Attempts have also been made to obtain specific precipitins from the gastric contents in cancer. Alfredo[10] prepared a rabbit serum by injecting cancer extracts, and this was tested against gastric juice *in vitro*. Cancer juices were found to precipitate the cancer serum, but apparently gastric juice in other conditions, *e.g.* ulcer, may produce a similar result.

In reviewing the methods described in this group, it will be noted that they are far more specific in character than those of the first two groups. Gluzinski's test is in a way intermediate between this group and the others, as it represents a *result* of the malignant growth on the secretory processes in the stomach, which, though not specific, is somewhat characteristic. Many possible sources of error surround the tryptophane test, and in early cases it is not infrequently negative. These tests may therefore be considered not sufficiently biological in character, while those depending on the production of anaphylactic shock are, if anything, *too* biological, and do not rest on a sufficient knowledge of the underlying principles of cellular physiology.

II. Tests applied to the Blood.

Passing on to the second main group of tests, two main sub-groups will have to be considered. The first sub-group consists of tests applied to the corpuscular part of the blood, and need not detain us for long. Two diagnostic points have been suggested as of value, namely the failure of digestion leucocytosis and the decrease in the hæmoglobin. The former is now recognised as unreliable, and the latter is not shown to occur sufficiently early to be of practical value.

We can therefore go on at once to consider the tests applied to the blood serum, and it is to be noted that the general criticism levelled against all these tests is that they are most likely indications that the originally local disease has become general, and therefore that the time at which diagnosis is essentially important has gone by. In some cases this may indeed be true, but it does not altogether follow that because alterations have been produced in the blood serum therefore actual metastasis must have taken place. Nor do we know,

I take it, how much " generalisation " is necessary to prevent successful surgical treatment of a primary growth. These tests, therefore, must be judged on their merits, with regard firstly to their clinical accuracy and secondly to their practical simplicity.

1. Macalister and Ross[11] have stated that the blood of cancer patients contains a substance or substances which excite pseudopodial activity in normal leucocytes, a reaction also observed with certain alkaloids. How early in a case of cancer this reaction may be expected to occur does not appear from their original communication ; and although they did not obtain any positive results in healthy persons or those suffering from diseases other than cancer, it is quite possible, as they themselves admit, that the phenomenon they describe is not really specific at all.

2. Two tests which may next be described seem to be connected with each other in some way, though not altogether running on parallel lines. The first of these is the reaction described by Brieger, Trebing and Marcus, and sometimes called Brieger's cachexia reaction. Normal blood serum contains a substance which is capable of inhibiting the proteolytic action of trypsin. The serum of guinea-pigs immunised to trypsin shows this property in a much higher degree. In certain diseases also the antitryptic power of the serum varies considerably. The test is usually carried out by dropping varying amounts of trypsin solution mixed with a constant amount of blood serum on to Petri dishes filled with a solid medium made from ox serum and incubating them. Where the trypsin has acted on the medium small depressions are found, and the antitryptic action of the serum to be tested is measured by the strength of dilution of trypsin it can inhibit. Normal blood serum apparently inhibits trypsin solution in 1 to 4 dilution. The antitryptic power is much decreased during digestion and often in diabetes ; it has also been found decreased in chronic Graves's disease, primary untreated syphilis, and in catarrhal jaundice. On the other hand, in pneumonia, it is increased up to the crisis, and then falls to normal as the physical signs disappear, and in lymphatic leukæmia and

various blood diseases it is also increased, but not always to any great extent. In a large number of cases of carcinoma the antitryptic power of the serum is considerably increased, thus the proportion may be 1:10 or 1:8 instead of the normal 1:4. Other cases of cancer give only a slight increase, such as might be found in normal persons. Now in some patients showing an increase in the antitryptic action of the serum the increase disappears after trypsin has been given by the mouth, whereas in others a still further increase occurs and is maintained. This latter reaction is taken to indicate a rather worse prognosis. Brieger considers that the increased antitryptic power of the serum is a measure of the cachexia induced by the disease. It is not specific for cancer, as it occurs in other chronic cachexias, but it may be of value in prognosis, and a negative result of the test is against the presence of cancer.

3. The next test is that described by Shaw-Mackenzie,[13] namely the increased power of activating the fat-splitting pancreatic ferment which is possessed by the serum of carcinomatous patients. According to his observations, human and animal serum itself has no lipoclastic or fat-splitting action, but is capable of activating pancreatic lipase. This activating power is much increased in carcinoma and in certain other diseases, such as diabetes and possibly tuberculosis. This reaction which, unlike the antitryptic action, has an accelerating and not an inhibiting effect, is also unlike it in that it persists after recovery or removal of the growth, whereas the antitryptic power sinks to normal under these conditions.

The lipase, according to Rosenheim's experiments, consists of two parts, which may be separated by filtering a glycerine extract of pancreas. The solid residue is inactive and the filtrate almost inactive, but mixed together they are active. The solid residue is thermolabile, and may be activated not only by the thermostable filtrate, but by normal blood serum and to a still greater extent by carcinomatous blood serum. The activity is measured by titration of the fatty acids produced. Shaw-Mackenzie believes that this reaction, together with the antitryptic, are strong evidence of the presence of

cancer, while their absence excludes it. It is, however, not yet clear whether these reactions are constantly present in the very early stages of the growth.

4. Freund and Kaminer[14] noted that normal serum contained a substance which dissolves cancer cells, whereas serum from patients with carcinoma failed to do so. This reaction, however, is not constant, and there are considerable difficulties in obtaining a suitable emulsion of cancer cells. Monakow found that sera of all cancer cases showed a destructive action on cancer cells in at least one-fifth of the cases. Other diseases give the reaction in at least 15 per cent. of the cases, and in three early cases of cancer it was only positive in one (Kraus, Graff and Ranzi).

5. Cancer serum has also been found in certain cases to dissolve normal red corpuscles, though not those of the patient.[15] This reaction is again not constant, and has been calculated to be present in only about 40 per cent. of the cancer serums. A similar reaction, though apparently to a lesser extent, has been shown to occur in tuberculosis and in other diseases. The hæmolytic action is destroyed by heating to 50 deg., but after heating the activity is restored by admixture of fresh normal serum. The reaction, therefore, appears to be due to a substance belonging to the class of bodies like the solid residue of pancreatic extract above described, that is to the amboceptors or immune bodies. It has been named isohæmolysis. A modification of this test, in which the hæmolysis is made to take place subcutaneously by injecting a suspension of normal corpuscles under the skin of the cancerous patient, was introduced by Elsberg and others in 1908. Various changes took place within a few hours at the site of injection, which were said to be characteristic of cancerous hæmolysis. The originators claimed 77 per cent. of positive results in cancer, but Risley, who repeated their experiments, only got 33.5 per cent. of positive results in cancer, while only 75 per cent. of non-malignant cases were negative. Moss investigated the natural hæmolysins, and found that though not present so generally as the agglutinins, they seemed to be governed by similar laws.

Considerable variation in agglutinative power of normal serum and in responsive reaction to agglutinins in red corpuscles occur in different individuals ; some sera contain no agglutinins, and some corpuscles never agglutinate. Other sera will only agglutinate certain corpuscles, and again, some corpuscles can only be agglutinated by certain sera. Gorham and Lissen have shown that these conditions also apply to a great extent to hæmolysis. By using Elsberg's method they obtained 60 per cent. positive results in cancer and 88 per cent. negative results in non-cancerous cases. The patients were tested with four different groups of corpuscles. They consider that the hæmolytic test is certainly not specific, and that the results obtained by test-tube experiments are quite different from those obtained by the subcutaneous method. Considerable importance attaches to the sort of corpuscles used, owing to the varying capacity for agglutination in the corpuscles of different individuals. They find positive reactions more significant than negative, but note that at present there is no evidence of the value of this test in doubtful or border-line cases.

6. The complement deviation method has also been extensively tried in cancer with somewhat confusing results. A large number of synthetic antigens have been tested, as well as various antigens prepared from malignant growths. The synthetic lipoids give equally strong reactions with malignant and non-malignant cases, and the various methods for preparing antigens from tumours or pancreatic extracts give bodies which are neither stable nor specific. In particular it has been found impossible to differentiate syphilis from cancer by this test.

7. The remaining test in this group is that known as the meiostagmin reaction.[17] Traube showed that the addition of a toxin to an anti-toxin caused a diminution of surface tension, and consequently that the size of a drop of the liquid was diminished and a given volume of the fluid would yield an increased number of drops. The instrument he devised for measuring the drops is called a stalagmometer, and consists of a finely-graduated pipette with a central bulb, holding about 8 c.c. The lower end forms a capillary tube, the extremity of

which is ground flat and has a diameter of about 7 mm. When full it contains a constant number (about 56) of drops of distilled water at 15° C., and all the drops, which can be counted as they fall from the ground base, are of equal size. Fractions can also be estimated. Ascoli first applied this principle to typhoid sera. The diluted serum was mixed with a fixed quantity of alcoholic extract of B. typhosus and the number of drops immediately counted. The preparation was then incubated for two hours, cooled, and the drops again counted. With very dilute antigens there was an increase of two or three drops, but no increase was obtained with normal sera nor with an antigen prepared from B. coli. Similar results were obtained in syphilis, and the reaction was then extended to cancer. The main difficulty in this test as applied to cancer is the preparation of the antigen, which appears to be a lipoid body. Several methods have been adopted from time to time, and it has been shown that only a certain proportion of cancerous tumours are suitable for preparing it, and it is not very stable. The most recent antigen is a methyl alcohol extract, which is titrated against normal serum, the weakest dilution which fails to give an increase of more than one drop with diluted normal serum being used. Stronger dilutions of the antigen are unreliable, because they give a positive reaction with normal sera.

As to the specific character of this reaction, it has been shown that a positive result can be obtained occasionally in other diseases, such as diabetes, tubercle, febrile and septic conditions, but it is not a cachexia reaction. This appears to be the most satisfactory of all the laboratory tests ; the main difficulty consists in obtaining a satisfactory antigen. Ascoli and Izar obtained a positive reaction in over 93 per cent. of cancer cases, Verson in 55 per cent., Stabilini in 100 per cent., Kelling in 47 per cent., Michaeli and Cattoretti in 87 per cent., and Stammler in 73 per cent. The last named also obtained a positive reaction in 20 per cent. of non-malignant cases. Tadesco obtained 93 per cent. positive in malignant disease and 12 per cent. positive or doubtful in other cases. Kraus and others got 92 per cent. positive in malignant cases.

Stammler has modified this test, and instead of using the drop reaction, relies on the formation of a precipitate when the antigen is incubated with the patient's serum. He obtained 83 per cent. positive results with cancer cases and 14 per cent. with other diseases.

III. Tests applied to the Urine.

I now pass on to consider those tests which are applied to the urine. These are all devised with a view to showing either that certain changes of metabolism have been set up by the cancerous process which can be recognised by the chemical character of the excretions, or that certain ferments are being excreted as a concomitant of the cancerous condition, which are not present to the same extent in health.

1. The first of the latter group is the presence of a reducing ferment in the urine capable of decolourising methylene blue.[18] This test has the advantage of extreme simplicity, for all that is necessary is to mix the urine with the ordinary solution of methylene blue and leave it to stand in a warm place for some hours. If the colour is destroyed a reductase is present and the test is positive. The disadvantage of this test is that it has been shown to be quite unreliable, as all cancer cases do not give a positive result and many other conditions do.

2. More complicated is the determination of the proteolytic ferment in the urine.[19] The method used consists of incubating the clear urine in various concentrations with a dilute solution of ricin, which has been precipitated with decinormal hydrochloric acid. However, the published results are contradictory, for two reasons—firstly, that different experimenters have adopted different methods; and secondly, that in many cases the clinical diagnosis has not been confirmed. In cases of simple achylia urinary pepsin may be low or high as compared with gastric pepsin, and in some cases of cancer it is present in large amounts in the urine. On the other hand, it may be absent completely in achylia at certain times. Some cancers also show complete absence of urinary pepsin. Thus, though pepsin in large amounts in the urine may point to cancer, the

results on the whole are too variable to be of clinical value, and it is especially in early cases that these variable results are obtained.

3. A large amount of work has recently been done on the urinary nitrogenous bodies in relation to cancer. It seems clear that under certain abnormal metabolic conditions there are variations in the form in which nitrogen is excreted, but so far the results do not seem sufficiently definite to be of great value.

The principal methods which have been suggested are—

(*a*) Precipitation with 5 per cent. phosphomolybdic acid. The urobilin must be removed by washing the precipitate with absolute alcohol. The occurrence of a precipitate is only suggestive of cancer, and only occurs in about half the cases.[20]

(*b*) Increase in the proportion of nitrogenous substances precipitated by alcohol. This is known as Salkowski's test, or the colloidal nitrogen test.[21] An increase in the proportion of colloidal nitrogen was considered diagnostic of cancer. Later, instead of alcohol, salts of the heavy metals, such as subacetate of lead or zinc chloride, were used as precipitants after removing the albumin with baryta mixture. Lower values are obtained by this method.

Confirmatory evidence of the value of these methods is wanting, the conclusions of other authors being summarised as follows :—

(i) Higher values of colloidal nitrogen are not specific for cancer. Not only do some cases give a low percentage of colloidal nitrogen, but there is a large series of cases, especially cirrhosis of the liver, diabetes, and infectious fevers, which give more or less constantly high figures.

(ii) With these exceptions it is true that cancer cases on an average give a higher percentage than healthy persons or non-cancerous cases.

(iii) The condition of nutrition, the food and the nitrogen balance influence the result of the test.

(*c*) A similar method is the estimation of the polypeptids in the urine.[22] The amino-acids are first estimated by formalin

titration ; the polypeptids are then split up by means of strong HCl, the acid removed, and the resulting amino-acids again estimated. The difference gives the amount of polypeptids. Although polypeptids on an average are increased in cancer, yet this method is open to the same criticism as the last.

(*d*) A peculiar group of polypeptids, namely the oxyproteinic acids,[23] have also been found to be increased in cancer. The method for their estimation by Solomon and Saxl depends on their sulphur content. The method is very elaborate, as the inorganic sulphur and ethereal sulphates have first to be removed, and the polypeptid sulphur then oxidised by perhydrol or sodium nitrite.

This reaction has now been applied to about 223 cases, with positive results in about 70 per cent. It does not appear to be due to cachexia ; it also occurs in other conditions (5 per cent.), but it seems to appear early in cancer cases. The elaborate technique is against its general adoption.

4. A reaction differing somewhat from those in the last group was described in 1910 by Royle.[24] She found that in cancer patients the ratio of the urinary phosphates to the uric acid was decreased, that is to say, that on a standard diet the excretion of phosphates decreased and the excretion of uric acid increased. As yet the results have not been confirmed on a large scale, but the reaction appears to be so similar to those previously described that it seems probable that it will be shown to be dependent on some condition frequent in cancer, but common to it and certain other disordered states of metabolism.

The examination of the urine cannot be said to have hitherto provided a satisfactory aid to the diagnosis of cancer. The methods are for the most part complicated, and the tests have in no case been shown to be pathognomonic. They appear to occur early in the disease, however, and to be dependent on metabolic changes other than mere cell destruction or cachexia.

As far as the results hitherto obtained go, a positive result from the meiostagmin reaction and the method of Solomon and Saxl for estimating the oxyproteinic sulphur would seem

to give a very strong presumption in favour of cancer, provided that certain other conditions can be otherwise excluded. On the negative side, an absence of increase in the antitryptic power of the serum and an increase in the lipoclastic accelerators is against the presence of malignant disease.

BIBLIOGRAPHY.

[1] Moore, Roaf and Kelly, *Biochem. J.*, 1906, i. 274, 297.
Idem, *Brit. M. J.*, 1906, ii. 213.
Boas, *Deutsche med. Wchnschr.*, 1911, No. 49.

[2] v. Aldor, *Wien. klin. Wchnschr.*, 1907, xx. 598.
A. J. Clark, *St. Barth. Hosp. Rep.*, 1910, xlv. 97.
Volkmann's *Sammlung*, Neue Folge, No. 387, 1906.

[3] Sigel, *Berl. klin. Wchnschr.*, 1904, xli. 299, 338.
Salomon, *Deutsche med. Wchnschr.*, 1903, No. 31.
Mathieu, *J. de Méd. et de Chir. prat.*, Sept. 10th, 1909.

[4] Mansell Moullin, *Lancet*, 1910, i. 417.

[5] Sigel, *loc. cit.*
Gluzinski, *Wien. Klin. Wchnschr.*, 1912, xxv. 553.
Alfredo, *Riforma Med.*, March 24th, 1908.

[6] Wolff and Junghans, *Berl. klin. Wchnschr.*, 1911, xlviii. 978.
Thiele, *Ibid.*, 1912, xlix. 544.
Ernstein, *Med. klin.*, March 24th, 1912, p. 484.

[7] Kuttner and Pulvermacher, *Berl. klin. Wchnschr.*, 1910, xlvii. 2057.
Ehrenberg, *Ibid.*, 1911, xlviii. 704.
Sigel and Alfredo, *loc. cit.*
Neubauer and Fischer, *Deutsche Arch. f. Klin. Med.*, 1909, xcvii. 499.
Lyle and Kober, *N. York M. J.*, 1910, xci. 1151.
Weinstein, *J. Am. M. Ass.*, 1910, lv. 1085.

[8] Schryver and Singer, *Quart. J. Med.*, 1912, vi. 71.

[9] Liveriato, *Centralbl. f. Bakteriol.*, 1910, lv. 510.
Pfeiffer and Finsterer, *Wien. klin. Wchnschr.*, 1909, xxii. 989.

[10] Alfredo, *loc. cit.*

[11] Macalister and Ross, *Proc. Roy. Soc. Med.* (Med. Sect.), 1908, ii. 50.

[12] Marcus, *Berl. klin. Wchnschr.*, 1908, xlv. 689.
Brieger and Trebing, *Ibid.*, p. 1041, 1349, 2260.
Hort, *Brit. M. J.*, 1909, ii. 966.

[13] Shaw-Mackenzie and Rosenheim, *Proc. Roy. Soc. Med.*, 1910, iii. (Pathol. Sect.) 168.
Shaw-Mackenzie, *Ibid.*, 1912, v. (Therap. and Pharmacol. Sect.), 152, 1912.

[14] Kraus, v. Graff and Ransi, *Wien. klin. Wchnschr.*, 1911, xxiv. No. 28.
Monakow, *München. med. Wchnschr.*, 1911, lviii. 2207.

[15] Elsberg, *J. Am. M. Ass.*, 1909, lii. 1036.
Elsberg, Neuhof and Geist, *Am. J. M. Sc.*, 1910, cxxxix. 264.
Crile, *J. Am. M. Ass.*, 1908, l. 1583, li. 2036, 1908.
Warfield, *Arch. of Int. Med., Chicago*, No. 15, 1911.
Simon, *J. Am. M. Ass.*, 1908, li. 915.
Gorham and Lisser, *Am. J. M. Sc.*, 1912, cxliv. 103.
See also *Brit. M. J.*, 1912, i. 1138 (references).

[16] *Brit. M. J.*, 1912, i. 1030 (references).

[17] Traube, *Pflüger's Archiv.*, 1908, No. 123, p. 419.
Ascoli, *München. med. Wchnschr.*, 1910, lvii. 62.
Ascoli and Izar, *Ibid.*, p. 403.
Bernstein and Simons, *Am. J. M. Sc.*, 1911, cxlii. [852] (references).
Brit. M. J., 1912, i. 1081 (references).
Leidi, *Berl. klin. Wchnschr.*, 1911, xlviii. 1716.
Verson, *Wien. klin. Wchnschr.*, 1910, xxiii. 1102.
Stabilini, *Berl. klin. Wchnschr.*, 1910, xlvii. 1498.
Kelling, *Wien. klin. Wchnschr.*, 1911, xxiv. No. 3.
Michaeli and Cattoretti, *Ibid.*, 1910, xxiii. 1555.
Stammler, *Munchen. med. Wchnschr.*, 1911, lviii. 1957.
Idem, *Arch. f. klin. Chir.*, 1911, xcvi. 158.
Tadesco, *Wien. klin. Wchnschr.*, 1909, xxii. 954.
Izar, *Ibid.*, 1912, No. 33.

[18] Fuhs and Lintz, *J. Am. M. Ass.*, 1911, lvi. 1882.
Verbrycke, *Med. Rec.*, 1911, lxxx. 876.

[19] Bieling, *Deutsche Arch. f. klin. Med.*, 1911, cii. 507.

[20] Ascoli and Grazia, *Berl. klin. Wchnschr.*, 1901, xxxviii. [1009] (references).
Ury and Lilienthal, *Arch. f. Verdauungskr.*, 1905. xi.
Von Aldor, *loc. cit.*

[21] Salkowski, *Berl. klin. Wchnschr.*, 1905, xlii. 1581.
Idem, *Ibid.*, 1910, xlvii. 533, 1746, 2297.
Mancini, *Deutsche Arch. f. klin. Med.*, 1911, ciii. 288.
Ischioka, *Med. Klin.*, 1911, vii. 1548.
Gross and Reh, *Ibid.*, 1911, vii. 778.
Kojo, *Ztschr. f. Physiol. Chem.*, 1911, lxxiii. 416.

[22] Sörensen, *Biochem. Ztschr.*, Bd. vii.
Frey and Gigon, *Ibid.*, Heft 304.
Henriques, *Ztschr. f. physiol. chem.*, Bd. lx.
Falk, Solomon and Saxl, *Med. Klin.*, 1910, vi. 510.

[23] Abderhalden and Pregl, *Ztschr. f. Physiol. Chem.*, Bd. xlvi., 1905.
Solomon and Saxl, *Wien. klin. Wchnschr.*, 1911, xxiv. 449.
Kaldeck, *Ibid.*, p. 1681.
Mazzitelli, *Riforma Med.*, July 27th, 1912.

[24] Royle, *Lancet*, 1910, ii. 449.

PAROXYSMAL ŒDEMA OF THE LUNGS.

BY

C. E. S. FLEMMING, M.R.C.S. Eng., L.R.C.P. Lond.

ACUTE œdema of the lungs, better called paroxysmal œdema, is a condition treated by the text-books in the most cavalier manner, though it is a very serious and probably a not very infrequent affection. In twenty-seven years' private practice

I have recognised four cases, and may possibly have overlooked others. I will give as concisely as possible an account of the signs and symptoms common to my cases.

The attack most often commences with some irritation in the throat, tickling and feeling of dryness with slight cough, and the patient has a vague fear of some serious happening. In later attacks this fear amounts almost to terror. He cannot, dare not, lie down; breathing at once becomes rapid, the face looks anxious and is pale. There is generally, but not always, some cyanosis with the pallor. The skin is cold and moist; there is a sense of suffocation; breathing is oppressed and urgent; cough grows more distressing, and there is some expectoration of frothy coagulable fluid, which is frequently blood-stained; the chest is rapidly filled with noisy râles; the pulse is small and quick; breathing becomes more distressing; expectoration at times becomes more profuse, but this is not by any means always the case. This condition continues for perhaps half an hour or an hour and a half. Gradually the breathing becomes deeper, easier and quieter, the pulse is fuller and less rapid, the skin resumes its normal colour and warmth; the patient, though exhausted, is thankful for the relief, but too nervous of a return of the symptoms to sleep soundly. Air now enters the chest freely, but râles persist over the greater part of one or both lungs. In one or at most two days all physical signs of the œdema have disappeared, and the attack is a thing of the past, to return, however, it may be in a week or two, it may be not for months. In one case the second attack was fatal; in another attacks recurred at irregular intervals for more than two years, the patient dying eventually of heart-failure following shingles.

The onset was nearly always at night, within two or three hours of bedtime. I have never been able to satisfy myself as to an exciting cause. An attack was generally attributed to one of those things that happen so frequently as to make them possibly mere coincidences, *e.g.* worry, excitement, a cold wind, a hot day, fatigue, some food that might not have agreed, being in a crowded room, or going upstairs. Of course, some such

event may have been a determining cause, but with one of my patients I was able to eliminate most of these causes. He had a lift to take him upstairs, his diet was most carefully regulated, he never went into a crowded room, but still the attacks recurred. I thought at first that avoiding the stairs had done good; certainly for six months after the lift was installed he kept well, but eventually the paroxysms returned.

Two of my patients were men, two women. The youngest was fifty-four, the others sixty-eight, seventy-three and seventy at the time of their death. The youngest was a woman who no doubt drank a good deal more than was good for her; she had a high tension pulse, a trace of albumin in her urine, and I thought suffered from pseudo-angina. I saw her during her first attack, which cleared off quickly. A month later she had another; and although the lungs cleared up quickly, her general condition was not good. She was weak and her pulse continued too fast. Ten days after her second attack I examined her chest; the lungs were then quite clear of adventitious sounds, and she seemed to be decidedly better. I had not left the house two minutes when a messenger ran after me. I returned at once, and found her struggling for breath, noisy râles all over the chest, with free expectoration of watery fluid. She became deeply cyanosed, and in two hours died apparently from heart failure.

The other female patient, aged seventy, was the hard-working wife of a labourer. She had had mitral incompetence for some six months, had marked thickening of her arteries, and no albumin in her urine. She had typical attacks at intervals of a few weeks, but after a few months became depressed and drowned herself.

A man of sixty-eight, whose physical signs were an apex beat displaced to the nipple line, thickened arteries, and a pulse tension of 180 had a good deal of dyspnœa and palpitation on exertion, and was a heavy smoker and eater, but temperate in his drink. He had a history of what I thought was an attack of angina. Two paroxysms of œdema occurred at an interval of about a month. I did not see him during the second seizure,

as he died before I could get to him, but from the account I received I had no doubt of its nature.

My longest case was a man of seventy-one. He had led a temperate and healthy life, but for two or three years had shown various evidences of arterial degeneration, two slight cerebral hemorrhages and rapidly-failing memory. His pulse tension was only 140, urine normal; he suffered from osteo-arthritis of one hip. For two years his attacks recurred at quite irregular intervals, and he died eventually of heart failure after an attack of shingles. In this case the attacks were followed by dulness over the lower half of the right chest, which persisted for some time, one week to two or three weeks; air appeared to enter less freely over this area, but there were very few râles.

I said that paroxysmal œdema was a better name than acute œdema, as it is necessary to distinguish these conditions from the acute œdema of pneumonia, influenza, advanced mitral disease, dilated heart and Bright's disease, often acute in onset but persisting.

It bears a curious resemblance to cardiac asthma in its mode of onset and in the general appearance of the patient during a paroxysm, but the state of the patient is much more alarming; there is serous effusion into the bronchi, and there is not necessarily other definite evidence of cardiac incompetence.

Various theories have been advanced as to the mechanism of an attack. The one that most appeals to me is that the left ventricle fails to contract properly, so that with every beat it is passing on less blood than the right, the consequence is that pulmonary vessels very rapidly become engorged. Professor Welch produced acute œdema in a rabbit's lungs by squeezing the left ventricle and so paralysing it.

This preliminary failure of the left ventricle becomes evident clinically in the pallor of the patient, the cold skin, the apprehension of death and the small rapid pulse, and the engorgement of the pulmonary vessels by the warning cough and the blood-stained serum that is early expectorated.

This alone does not explain the onset or the cause of an

attack. Whether the ventricle fails from some undue strain or from disease of some branch of the coronary artery, or some affection of the auriculo-ventricular bundle, whether the œdema that so rapidly follows the congestion is due to disease of the vessel walls, of the bronchi or alveoli, or to an unhealthy blood state, or to both, there is not as yet enough evidence to prove. It may be that the recurrence of the seizure is due to an accumulating toxin. It may be that rapid breathing, which accompanies the nervous onset at the moment the left ventricle fails, produces a state of deficient carbon dioxide in the blood, a state which would favour the rapid exudation of fluid from the plasma to the tissues or the bronchial tubes. This exuding fluid would prevent the further exhalation of carbon dioxide and soon cause it to be increased in quantity in the blood, and so stimulate the respiratory centre to increased respiration ; but now, the lungs being water-logged, this increased respiration is of little avail, the carbon dioxide cannot escape, and the oxygen cannot be absorbed. I only suggest this hypothesis for the consideration of those better able to criticise. My aim is merely to present a clinical record. To complete this I must say a few words about treatment. Nitrites in any form I found useless ; and if the theory of failing left ventricle is correct, this is what one would expect, for the nitrite would not alter the ratio between the work of the right and left ventricles. Large hot fomentations no doubt gave some relief ; they were soothing, but I do not think that they cut short the attack. I found no evidence of the value of stimulants. Oxygen given freely—it must be freely—gave decided relief, and I think shortened the attack ; but nothing to my mind gave so much relief as a hypodermic injection of morphia and atropine. I have only used it in one case, but there it certainly acted like a charm when given soon enough. Since using it for this patient I have regretted that I did not use it before. Whether it acts by quieting the patient's nervous state or by direct action on the heart, I will not venture to say.

NOTES ON TWO CASES OF URINARY CALCULUS.

BY

C. A. MOORE, M.S. Lond., Ch.M. Bristol, F.R.C.S.,
Assistant Surgeon, Bristol General Hospital.

THE condition of calculus in the urinary passages is always an interesting one, and there are special points about both the cases described below.

Case 1.—F. P., male, age 38, admitted July 19th. Complains of lumbar pain and increased frequency of micturition. The previous history contained nothing of importance beyond the fact that the patient had been in hospital in India for malaria while he was serving in the army. The present illness began three months ago with frequency of micturition every two hours by day and about three times at night. Three weeks later patient began to suffer from pain across the loins behind, below the last rib on each side, much worse on the right side. The pain was a dull ache, not of a colicky nature, and did not pass down towards the groin or testicle. A month before admission micturition became painful, the pain being in the hypogastrium and along the urethra, especially severe towards the end of micturition. Three weeks before admission blood appeared in the urine for the first time. It came on suddenly, and at first the urine was deeply blood-stained—the urine being almost pure blood, according to the patient's account. It gradually cleared until it was only faintly stained, but generally a few drops of blood passed towards the end of micturition. Just before admission the blood ceased, but the pain and increased frequency continued. No "gravel" was ever noticed in the urine. On admission the patient was pale and rather thin, but looked fairly well. Nothing abnormal was revealed by examination of the chest. On examining the abdomen there was a markedly tender spot just internal to the left anterior superior iliac spine. The kidneys could not be palpated, but there was marked tenderness when pressure was made in the loins behind. The lumbar spine was held somewhat rigid on stooping, but no pain was elicited on "crowding" of the vertebræ. Cystoscopy and rectal examination both failed to reveal any abnormality. The urine was acid, and contained a good deal of pus, but no blood corpuscles, and examination for tubercle bacilli was negative. The temperature chart showed curious elevations of temperature. At intervals of roughly a

week, between July 19th and September 2nd, the temperature rose to 102 to 104 deg., with a rigor on several occasions, but no urinary or other symptoms accompanied these elevations of temperature. Skiagrams were taken, and clearly showed a small shadow in a position corresponding to the pelvic part of the left ureter, but none elsewhere. A second skiagram taken a fortnight later confirmed the appearance seen in the first.

In view of the doubt as to the side affected, I decided, at Mr. Morton's suggestion, to explore both urinary tracts through a median laparotomy incision below the umbilicus. The right kidney was distinctly larger than usual, but felt perfectly normal otherwise, nor could anything resembling a calculus be felt along the course of the ureter. The left kidney was distinctly smaller than usual, perhaps three inches long or a little less, but in consistence it felt perfectly normal. The abdominal part of the ureter contained nothing, but in the course of the pelvic ureter, and just outside the bladder, a small, hard mass could be easily felt, corresponding in position to the shadow seen in the skiagrams. This appeared to confirm the diagnosis of left ureteral calculus. The mid-line incision was therefore closed, and an extraperitoneal ilio-inguinal incision made. The foreign body which had been felt proved to be a calculus in the ureter, immediately outside the bladder wall. The ureter was incised and the calculus extracted. The incision was so inaccessible that no attempt was made to close it, and the wound was stitched up with a drainage tube passing down to the wound in the ureter. The calculus was the size of a large cherry-stone, partially divided into two by a groove. It weighed about nine grains. Urine was discharged along the track of the drainage tube for nearly three weeks; this then ceased, and the wound became soundly healed. After the operation the patient expressed himself as quite free from pain in the right loin and elsewhere, and was discharged looking and feeling quite well.

A few months later I saw the patient again. He was complaining of dull pain in the loins and some increased frequency of micturition. The wounds were soundly healed, and a skiagram showed no sign of any calculus. Examination of the urine, however, showed it to be swarming with bacillus coli, and a course of autogenous vaccines was given. This failed to entirely clear the urine of the organism, but caused considerable relief of his symptoms, although he still complained of some hypogastric pain when I last saw him some months ago.

The chief interest of this case centres round the difficulty of diagnosis. Although the chief pain was in the right loin, the only calculus discovered was low down in the left ureter.

Some years ago a theory, originated by Thornton and Guyon,

and supported by other surgeons, was much in vogue, the so-called reno-renal reflex. By this was meant a reflex pain caused in a healthy kidney by the presence of a calculus in its fellow. The possibility of this was denied by Morris, who held that pain in a healthy kidney never occurred except as the accompaniment of more severe pain in its fellow. At first sight reno-renal reflex pain seems a convenient explanation of the pain in the right kidney in the case described above. The right kidney appeared healthy to the hand in the abdomen, nor did a skiagram show any sign of calculus in it. Moreover, the pain was apparently caused by the stone in the left ureter, since all symptoms temporarily subsided with the removal of the calculus from the opposite ureter. However, the recurrence of slighter symptoms on that side on the onset, later, of *B. coli* bacilluria, obscures the issue, since obviously the pain might have been due to a *B. coli* pyelitis. The possibility of a reno-renal reflex pain cannot be lightly put on one side, since it is a well-known fact that a stone in the upper urinary tract may cause a reno-vesical reflex, with hypogastric and perineal pain and dysuria, when the bladder itself is perfectly healthy.

The only way to clear up the diagnosis seemed to be to explore both tracts through an abdominal wound. Although the shadow of the ureteral calculus was quite definite in all the skiagrams, one knows from experience that there are fallacies—due especially to the presence of phleboliths in the pelvic veins—to be guarded against in the interpretations of such skiagrams.

The more or less regular attacks of pyrexia, sometimes accompanied by a rigor, were another puzzling feature of the case, and it was never cleared up as to whether they were really due to the old malarial infection or not. Inquiry from the patient since he left the hospital showed that they have not occurred since, so they were presumably due to the urinary condition, although the last occurred some days after the operation.

Case 2.—E. F., male, age 28, admitted September 2nd, 1912. Since fourteen years of age patient suffered from pain in the left

loin and groin, for which a varicocele operation was performed when he was sixteen, but it gave no relief. When aged nineteen his left kidney was explored in the Gloucester Infirmary and a stone removed. His symptoms were entirely relieved, and for eight years he remained free from pain, but it began to return about twelve months ago, and had been increasing in severity for the last three months before admission.

The pain began in the left hypochondrium and passed down into the groin and testicle of the same side. It was made much worse by jolting, and came on in agonising attacks of colic, accompanied by vomiting. Frequency of micturition had never been increased, nor did the pain cause any strangury; the urine had often contained a white deposit, but never blood.

Except for some pallor, the man looked well. The chest was normal, and nothing unusual could be felt in the abdomen beyond some tenderness in the left loin, where there was a scar in the usual position for lumbar nephrotomy. The urine was of sp. gr. 1015, acid, contained urates and a trace of albumin, but no blood or pus. A skiagram clearly showed the shadow of a large stone in the left kidney, but none elsewhere. The patient informed me that he was told that he nearly died of shock and hemorrhage after the first operation. When told that the scarring left by the first operation would probably render a second more difficult, and therefore more dangerous, he expressed himself willing to take any risk necessary to relieve him from his miserable plight.

The kidney was cut down upon through the old scar. It was quite fixed, and could not be moved, much less delivered, on to the surface, the upper half being buried in dense scar-tissue. A large calculus could be clearly felt through the thinned cortex of the lower pole. An incision was made through this on to the stone, and an attempt made to extract it with forceps. This failed owing to the small opening, and the stone being wedged in the remaining kidney substance and scar-tissue. The opening was therefore made as large as possible, the stone grasped with the fingers, and torn out by force. Fairly smart hemorrhage followed from the torn cortex. It was too inaccessible to be controlled by sutures, but was easily checked by firm packing with gauze. The man lost a fair amount of blood, but his pulse never reached 130, and his condition caused no anxiety. Two days later the gauze was removed and the wound sutured, no hemorrhage ensuing. He left the hospital on September 26th, completely relieved of all his symptoms. The stone consisted of uric acid and weighed 2¾ ounces.

The interest in this case lies in the fact that it was apparently a case of recurrence of stone after a previous removal. Apparently it was not due to a piece having been left behind after the

previous operation, since the patient was entirely free from any symptoms for eight years.

While recurrence of a stone in the bladder not uncommonly follows removal, even by lithotomy, it appears to be a very rare thing for a kidney-stone to recur. A careful search through all the standard works on the subject yields only one reference to the possibility of such an occurrence. Freyer reports that in sixty-six operations for stone in the kidney five of them were for recurrences, and three of the original operations were performed by himself.

In the somewhat analogous case of gall-stones, experience proves how uncommon it is for stones to accumulate in a gall-bladder which has been once emptied by operation, but the reason for this immunity is not easy to understand. Even granted that it is possible by special dieting or drugs to prevent the formation of calculi in the kidney, it is unlikely that in hospital cases, at any rate, any such prophylactic treatment is ever systematically undertaken. It is still less likely that a hospital patient, once rid of his symptoms by an operation, would ever submit to treatment designed to prevent a recurrence.

URINARY PRODUCTS OF INTESTINAL INTOXICATION.[1]

BY

G. HELY-HUTCHINSON ALMOND, M.A., B.M., B.Ch. Oxon.

Hon. Pathologist to the Royal Mineral Water Hospital, Bath.

I PROPOSE to discuss the methods, the uses and the limitations of urinary analysis as an index of the excessive or perverted bacterial activity of the intestinal tract.

During the process of normal digestion the food is split up

[1] A paper read before the Bath and Bristol Branch of the British Medical Association on April 30th, 1913.

into simpler molecules, which can be readily absorbed by the intestinal mucosa, and which are there, in the liver, or in some other organ, prepared for absorption into the complex activity of protoplasm. Many of these products are in themselves poisons, and have to be re-combined or re-synthesised into harmless products. For this protective action the liver is mainly responsible, and as we shall presently see, it also takes a large part in detoxicating the products of bacterial energy.

The necessity of bacterial life in the intestine has been a matter of much controversy; but we do know that polar animals can exist and thrive with an alimentary canal that is practically sterile. Where germs freely exist, however, as they do in all other climates, the presence of bacteria in the intestine seems on the whole to be an advantage, for (under ordinary conditions) certain groups of organisms become immune to us and we to them, and they do their best to keep out the wild intruder by making his existence difficult. Under certain conditions, however, the flora may become abnormal, and when this occurs various forms of intoxication may result.

For the subject we have in view the bacterial flora may be roughly divided into two classes—those which mainly attack and decompose carbohydrates, and those that mainly attack proteins or their hydrolysed products. The action of the former class is called fermentation, of the latter putrefaction.

During the process of excessive *fermentation* abnormal quantities of alcohol and of lactic, acetic, butyric, formic or oxalic acid may be formed, one of the products usually predominating. The acidity of the urine may be increased, as the acids if not oxidised combine with sodium and potassium, which deprives the phosphoric and sulphuric acid of some of their bases; but if excessive carbonic acid is at the same time formed in the gut, this tendency is antagonised, and the urine may even become alkaline. If the formation is due to an excessive or mainly carbohydrate dietary micturition is apt to become more frequent and the urine is increased in amount, unless diarrhœa accompanies it, when it will become more concentrated and more acid.

Gastric fermentation accompanied by a large carbohydrate dietary not infrequently leads to increased tissue waste, with a consequent increase in the uric acid output.

Alcohol and the organic acids themselves are, however, not found in any but minute traces in the urine, with the exception of oxalic acid, for they are readily oxidised in the body. Oxalates, if not due to excess of spinach, rhubarb, etc., denote in any but minute traces a pathological condition, and indicate gastro-intestinal fermentation. The condition has been produced experimentally by Baldwin,[1] who fed dogs with large quantities of cane sugar or glucose and meat, substances that in themselves contain no oxalic acid. Excessive gastritis with fermentation and pronounced oxaluria was the result. Oxalic acid was also produced in considerable amount by inoculating a medium of sugar and albumoses with material from the gastric contents. Gastric dilation, besides lending itself to oxaluria, may cause an acidosis, which, though mild, is very persistent.

The measure of acidosis can be made in a very few minutes in the following way:—20 c.c. of urine to which about a gram of potassium oxalate has been added are neutralised with $\frac{N}{10}$ NaOH, using phenolphthalein as an indicator. The mixture is then treated with 5 c.c. of formalin neutralised in the same way. The latter forms a compound with the ammonia, which is combined with the acids present, and thus the acids are liberated. Titration again with $\frac{N}{10}$ NaOH will indicate the amount of acidosis, and calculations will give the amount per cent. either in the terms of acid or of ammonia present.

The graver forms of acidosis do not, however, enter into our subject as they are metabolic disturbances rather than intestinal.

Putrefaction, or the excessive breaking-down of protein, is the result mainly of anaerobic bacteria. The intestinal canal, which may be regarded as a long oxygen-free or anaerobic tube, lends itself readily to this type of organism.

The hydrolysis produced by the digestive juices and the protein-splitting of bacteria follow very similar lines of cleavage, the similarity being due to the fact that the simple protein molecule is a large complex of amino-acids united together by

their basic and acid groupings. The chemical action of the proteolytic enzymes of the intestine hydrolyses the complex protein food into albumoses and peptones, and peptone being soluble is rapidly absorbed. *In vitro* further splitting to amino-acids takes place, but this does not occur to a large extent in the living organism.

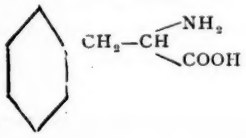
The action of the proteolytic bacteria, on the other hand, is a process of reduction. Oxygen is necessary for bacterial activity; and so, although the lines of cleavage are similar, the resulting products are different. Cleavage is extended further, and many substances useless for protoplasmic absorption are formed, many of them being actual poisons.

The amino-acids that enter into the composition of a protein molecule belong to three main groups. Firstly, we get the amino-acids of the fatty acid series. These contain one or two amino-acid groupings, the latter being known as the hexone bases. Secondly, there are the amino-acids of the aromatic series; and thirdly, there is cystin, a diamino-acid containing sulphur.

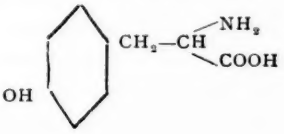
When proteolysis is excessive the fatty acid series are to a large extent broken up to the simple constituents of CO_2, H_2O, NH_3, etc. From the CO_2 and NH_3 carbonate of ammonia is formed, which is synthesised into urea in the liver. The higher compounds are, however, resynthesised by the absorptive epithelium into bodies useful for protein formation; so that as far as the fatty acid series of amino-acids are concerned, we have no urinary index of the condition, except in cases of grave hepatic inadequacy. With the aromatics, however, the case is different, and in many cases they act as a valuable guide as to the amount of putrefaction occurring.

The chief aromatic constituents of the protein molecule are phenylalanin, tyrosin and tryptophane.

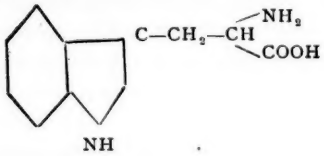
Phenylalanin.



Tyrosin.



Tryptophane.



These substances do not appear in the urine under ordinary circumstances, tyrosin does so only in cases of acute yellow atrophy and allied conditions, and once absorbed either as such or combined in protein, they become completely oxidised in the tissues, the benzene ring being split open. It is only in alkaptonurics that this does not occur, and under these circumstances the phenylalanin and tyrosin radicles appear in the urine as uroleucic and homogentisic acids.

When, however, intestinal putrefaction occurs, a more profound hydrolysis is associated with reduction, and bodies are formed in the gut and absorbed by the mucosa which are either protoplasmic poisons or substances which cannot be assimilated or made use of by the organism. These substances, if poisonous, are neutralised in the liver, and to a small extent in the tissues besides, to harmless aromatic sulphates or glycuronates, and are then excreted along with the non-toxic ones by the kidneys. As a general rule the greater portion of the aromatic products of putrefaction appear in the urine as sulphates.

Sulphates occur in the urine in two forms, the preformed or neutral sulphates and the ethereal. The former are principally formed from the products of tissue metabolism, and are practically proportioned to the nitrogenous output; the latter are usually almost entirely the products of intestinal putrefaction. Hence, if a quantitative estimate is made of both the preformed and the aromatic sulphates, and the ratio of the two is compared, an approximate estimate can be made of metabolism in general and of the extent of putrefaction, and the ratio which they bear to one another.

In health the daily output of the ethereal sulphates is from

.1 to .3 gram, reckoned as sulphuric acid, and the neutral sulphates from 1 to 3 grams; the ratio of the two is $\frac{1}{10}$ to $\frac{1}{16}$. In disease involving putrefaction the ratio may rise to $\frac{1}{8}$ or $\frac{1}{7}$ and even to $\frac{1}{1}$, or to a quotient greater than 1.

Quantitative Estimation of Sulphates in Urine.

Total sulphates, preformed and ethereal—

10 c.c. of urine (accurate)
10 c.c. of pure HCl.
20 c.c. of distilled water

are put into a flask and boiled gently for half an hour. A funnel is placed in the neck of the flask to act as a condenser. The mixture is then cooled and 10 c.c. of 5 per cent. $BaCl_2$ is added. The barium sulphate is then treated and weighed as below.

Inorganic sulphates—

10 c.c. of urine (accurate)
10 c.c. of pure HCl.
20 c.c. of distilled water
10 c.c. of 5 per cent. $BaCl_2$

are put into a flask and the mixture is then allowed to stand for half an hour.

A small Gooch crucible is meanwhile taken, and a watery mixture of refined asbestos is placed inside. The crucible is placed on the water pump to express the water, so that an even film of asbestos lies on the surface of the perforations. The crucible is then carefully dried, gradually heated, then placed in a blow-pipe flame, and then into a dessicator. After allowing to cool it is carefully weighed. The total sulphates are now filtered through it on a water pump, care being taken always to have the crucible at least half full of fluid until the end. Any remaining traces of sediment are washed out of the flask and added.

The crucible is then dried, warmed, and placed under the blow flame as before, care being taken not to allow the flame to play on the inner surface of the crucible. After cooling it is weighed as before, and the difference in weight gives the total sulphates.

The inorganic sulphates are now treated in the same manner, the same crucible being used.

By subtracting the inorganic sulphates from the total the weight of the organic sulphates can be determined.

Example.

Weight of crucible .. .. 13.927 grams
Weight on addition of total sulphates .. .. .. 14.002 ,, } Difference=0.075
Weight on addition of inorganic sulphates .. 14.069 ,, Difference=0.067

Total sulphates=0.075 gram. in 10 c.c. of urine.
=0.75 per cent.
Inorganic ,, =0.067 gram. in 10 c.c. of urine.
=0.67 per cent.
Organic ,, =0.008
=0.08 per cent.

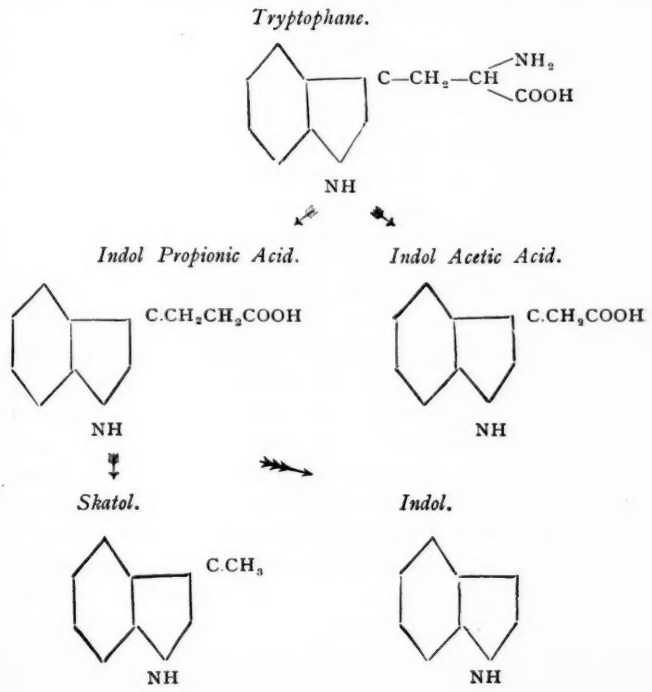
$$\text{Index} = \frac{\text{Organic}}{\text{Inorganic}} = \frac{0.08}{0.67} = \frac{1}{8}$$

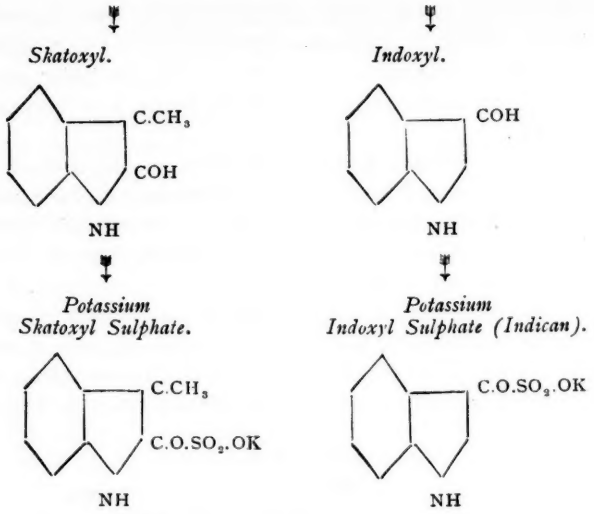
If total urine for 24 hours=1,000 c.c.
Organic sulphates=0.08 per cent.=0.08 × 10=0.8 gram.
Reckoned as barium sulphate=0.33 gram. H_2SO_4.
Inorganic sulphates=0.67 per cent.=0.67 × 10=6.7 grams.
Reckoned as barium sulphate=2.76 grams. H_2SO_4.

The chief aromatics found in the urine are indican or indoxyl sulphate of potash, skatoxyl sulphate of potash, indol acetic acid—all derived from tryptophane—and the phenol and cresol sulphates—derived in the most part probably from tyrosin. In some cases glycuronates are found, especially if the supply of sulphuric acid runs short. The phenol and cresol derivatives are usually found in the urine only in minute quantities. They are usually increased when there is a general increase in the ethereal sulphates, such as occurs in certain cases of intestinal obstruction and peritonitis, also in anæmias, diabetes, typhoid and general cachectic conditions. Occasionally the quantity is increased in proportionally much larger amount, but no special significance has so far been attached to this. If present in large quantity they will react to Millon's reagent applied directly to the urine.

The tryptophane derivatives have been more carefully studied, as they lend themselves peculiarly to colour reactions, and in many cases some shrewd idea of the particular nature and the type of organism that is causing the putrefaction can be obtained .

The two primary katabolic products of tryptophane on undergoing bacterial cleavage are indol-propionic acid and indol-acetic acid. The former is unstable, and from it are derived indol and skatol, which are in part absorbed, held up, chiefly by the liver, and after hydration to indoxyl and skatoxyl are combined with sulphuric acid, and are then excreted in the urine as the potassium salts, indican and skatoxyl sulphate of soda. Indol-acetic acid is, on the other hand, much more stable, and as we shall presently see is excreted by the kidneys unchanged.





The best-known tests for indican consist in treating it with some oxidising agent in acid solution and producing a deposit of indigo blue. The simplest way of performing the test is as follows :—Put about 5 c.c. of urine in a test tube and about 3 c.c. of HCl in another. Slant the acid HCl test tube, and place in the mouth of it a grain or two of bleaching powder. Now run the HCl gently over the bleaching powder into the test tube with urine. In a short time a well-marked blue ring will be formed if indican is present. As indigo is most insoluble in water and fairly soluble in chloroform it will be all taken up by chloroform if 2 or 3 c.c. are now added. The intensity of the blue is a rough indication of the quantity of indican present. Strauss' test is very similar, and consists in first acidifying the urine with lead acetate, filtering and adding equal quantities of HCl with a trace of ferric chloride in it and then chloroform.

This is the method that I am using for the quantitative estimation of indican :—

20 c.c. of urine are acidified with 5 c.c. of weak solution of lead acetate. 20 per cent. is advised, but I find this sometimes

too strong. 10 c.c. of this, equivalent to 8 c.c. of urine, are treated with 10 c.c. of Obermeyer's solution of HCl. and ferric chloride, and with about 5 c.c. of chloroform are put into a separator. The mixture is well shaken two or three times and the chloroform allowed to settle, and then run off. More chloroform is added and separated, and the process is repeated two or three times until the chloroform remains uncoloured. After noting the quantity of the chloroform extract, a measured quantity is placed in the Veley's colorimeter and measured against a chloroform extract of known strength. From this a twenty-four hours' estimation of indican can be made.

Herter's classification[2] of the main types of intestinal putrefaction has now been generally accepted. These are the indolic, the saccharo-butyric, and the combined indolic and saccharo-butyric. The classification is a generalisation only, and should be used as a starting-ground for investigation rather than as a finality in classification, a matter he would have been the first to emphasise. However, his *indolic* type is based on the fact that a large quantity of indol is formed in the intestine and a more or less proportional quantity of indican is found in the urine.

Indol is produced in quantity either through the ascent of the colon bacilli into the small intestine, where they have free access to the peptones, or through the descent of native proteids to the large intestine, where they are attacked by the combined action of the putrefactive anaerobes and the colon bacilli, and any factors that produce either of these conditions are such as lead to pronounced indicanuria.

From what I have said it can readily be gathered that excess of or indigestible forms of protein are more likely to lead to indicanuria than moderation and the taking of proteins that are rapidly absorbed.

Indicanuria in youth can generally be easily treated by dietary and mode of life ; moreover, its effects are not injurious, as the margin of compensation by oxidation in the tissues is large. With age this is not so easy, nor is the compensatory mechanism so perfect. Prolonged indicanuria, especially if associated with

the saccharo-butyric type of putrefaction, frequently leads to intractable neurasthenia. Lee [3] has proved experimentally that both indol and skatol have a fatiguing effect on muscle, a fact which may have some significance in this respect; but it is, on the other hand, quite possible for debility to be the cause of indicanuria. If the alimentary secretions are for any reason feeble, proteolysis, as will be evident from what I have already said, will become more marked, for there will be more pabulum for the bacteria to deal with. Constipation may or may not lead to increased production. If the lodgment is well down in the sigmoid indican need not necessarily be increased unless it leads to stasis higher up.

The measure of indican is not always proportional to the amount of putrefaction or to the quantity of ethereal sulphates. As I have indicated, the phenol derivatives may at times be excessive. Indol-acetic acid may also to a large extent take its place, for it is rare to find both indican and indol-acetic acid marked in the same person, so that in cases of marked indol-aceturia the reliability of the indican test is impaired.

The test [4] for indol-acetic acid in the urine depends on two things—the presence of a nitrite in small or minute quantities and the presence of HCl. If the urine has stood for twelve hours or so nitrites are usually present—the product of nitrifying bacteria.

Strong HCl. is added to an equal quantity of urine, and a rose-red colour should appear. If it does not, the addition of a drop or two of .2 per cent. potassium nitrite will bring it out. Should the presence of indigo obscure the reaction, both the indigo blue and the indigo red can be readily extracted with chloroform. The supernatant fluid can than be pipetted off and treated with amyl alcohol, which will take up the indol-acetic compound urorosein, as it is called. The precise nature of the reaction is not known.

There are, however, other ethereal salts that on the addition of HCl. form a red body, notably skatol red. They can both be extracted with amyl alcohol, and both have an identical spectroscopic band between D and E. They differ, in that urorosein has

a tint which is rose colour and is lighter and brighter than skatol red, which is redder. Urorosein is readily soluble in water, skatol red little so. The former is insoluble in ether and chloroform, the latter somewhat soluble. Administration of skatol to a dog will produce the skatol red reaction in urine and not the urorosein. The colouring matters derived from bile pigment may also give a somewhat similar HCl. reaction, but they can be thrown out by precipitation with lead acetate in dilute solution.

The significance of indolaceturia is not yet known, nor why it should supplant the indican and *vice versa*. In testing for ethereal bodies it should never be neglected. The clinical states that it is chiefly associated with are diabetes, typhoid fever, and chronic enteritis. It is marked in certain cases of jaundice and pulmonary tuberculosis. It has been noted in the vomiting of pregnancy, and it is generally present if there is any intestinal putrefaction.

One of the most marked cases described was of a boy of seven, whose skeletal and muscular development was severely retarded, and whose abdomen was markedly protuberant from long-standing distension with gases.

The paradimethylamidobenzaldehyde[5] reactions with the tryptophane derivatives are full of interest, but our knowledge of them is far from complete. Added to urine with HCl, with or without the aid of heat, a cherry-red colour is formed, which on the addition of a weak alkali to neutralise turns into a beautiful carmine. If amyl alcohol is now added, it takes up nearly the whole of the colouring matter. This reaction is influenced in several different ways, so that its full significance is not at present completely understood. It depends partly on the presence of urobilinogen. It is augmented by the feeding with or injection of skatol, or by the presence of much skatol in the fæces and *vice versa*. It is uninfluenced by administration of indol. Feeding with red meat increases it, but previous extraction of all the colouring matter diminishes.

Herter observed that the reaction corresponds in its intensity with the urorosein reaction after skatol administration, and he

was disposed to think that the absorption of skatol from the intestine is a common cause of the aldehyde reaction.

An exact knowledge of the chemical processes produced in our alimentary tract, of the influence which the products absorbed have on our system, how they aid in causing disease and senility, are subjects of the utmost importance to our profession. It may be many years before we can gauge their true significance, but I feel convinced that a more regular examination of the urine for the bacterial by-products of the protein molecule will go some way in hastening the unravelling of these knotty problems.

REFERENCES.

[1] *J. Exper. Med.*, 1900, v. 617.
[2] "Common Bacterial Infections of the Digestive Tract," 1907, p. 278.
[3] *Ibid.*, p. 255.
[4] *J. Biol. Chem.*, 1908, iv. 101, 239.
J. Am. M. Ass., 1908, l. 1959.
[5] *J. Biol. Chem.*, 1906, i. 415.

General.

Herter, "Common Bacterial Infections of the Digestive Tract," 1907.
Herter, "Lectures on Chemical Pathology," 1902.
Wells, "Chemical Pathology," 1907.

SOME RECENT CASES OF EPIDEMIC CEREBRO-SPINAL MENINGITIS.

BY

J. R. CHARLES, M.A., M.D. Cantab., F.R.C.P.,
Physician to the Bristol Royal Infirmary.

THE following cases of meningococcic cerebro-spinal meningitis are described because there can be little doubt that at the present time there is a small epidemic of this disease in Bristol; and further, the symptoms and signs, especially in the early stages, are by no means easy to elucidate without the use of special methods, such as that of lumbar puncture.

In the three cases here described Flexner's serum has been used. In the first case it appeared to exert a marvellously beneficial effect, the patient being rapidly cured, even though he was admitted in a comatose condition. Whether this was the effect of the serum, or merely due to coincidence, it is difficult to say, especially as urotropin was also freely given, as it was in the other cases.

One patient was the wife of one of the others, but whether she derived her infection directly from her husband or from a common source it is impossible to say. She sat, however, close by his side during his last illness.

Case 1.—L. H. T., aged 16, a male, was admitted to the Bristol Royal Infirmary on November 23rd, 1912. He had suffered from a headache for a fortnight, and had given up his work two days before admission. During that day he became unconscious, and remained so until admission. He was then semi-conscious, and could be roused by a stimulus. He had extensive herpes labialis, was irritable, resented being touched, and lay curled up in bed. Temp. 97.8; pulse 70; resp. 24. His temperature during his stay in the Infirmary only reached 100 deg. F. on three occasions, and was as a rule either about normal or slightly subnormal. His pupils were equal and reacted to light, and on accommodation; there was definite optic neuritis in the right eye and some swelling of the left disc; no squint or nystagmus. The other cranial nerves were normal. No paresis of any of the muscles of the limbs could be detected. The deep reflexes were normal, and the plantars gave a flexor response on each side. There was very slight rigidity of the neck and a doubtful Kernig's sign. On the 25th a lumbar puncture was performed. The cerebro-spinal fluid was under increased pressure, and consisted in appearance of almost pure pus, in which the meningococcus was demonstrated by Professor Walker Hall. On this day there was more rigidity of the neck, Kernig's sign was present, and also a divergent strabismus. The mental condition remained as before. After an equivalent amount of cerebro-spinal fluid had been removed, 15 c.c. of Flexner's anti-meningococcic serum were injected into the cerebro-spinal canal. The following day the patient was much less drowsy and could answer questions. His squint had gone, and he complained of no headache. The other physical signs remained much the same, except that the plantar response was extensor on both sides. 15 c.c. more serum were given subcutaneously. Improvement continued, and he was much brighter mentally. The cerebro-spinal fluid was, however, still very

turbid, though fewer organisms were present. Another 20 c.c. of serum were given intra-spinally. On the 27th improvement continued. The cerebro-spinal fluid was nearly clear, and 20 c.c. of serum were again given intra-spinally. On the 29th his mental condition appeared normal; Kernig's sign, Babinski's sign, the strabismus and head retraction had all disappeared, and the swelling of the discs was subsiding.

Following this he made a rapid and uninterrupted recovery, and was discharged cured on December 21st, when the only thing which could be detected as abnormal in his condition was a little patch of lymph round the right disc. He has been seen on several occasions since, and remains perfectly well. It should also be added that during the acute stage of the disease 10 grains of urotropin were given every four hours.

Case 2.—H. G., aged 45, a male, was admitted on April 17th, 1913, to the Bristol Royal Infirmary (under the care of Dr. Prowse, who has very kindly given permission for the publication of the case), complaining of a pain in his head, neck and legs. He had been ill for four days, the illness beginning with a shivering attack. He became unconscious on the evening of the 16th, and was unconscious on admission. He also had a crop of herpes labialis. His breathing was stertorous. Temp. 100.4; pulse 92; resp. 32. His urine was acid, 1025, and contained a trace of albumen and some sugar. There was no acetone or diacetic acid. Nothing abnormal was found in the heart or abdomen. In the lungs there was a little bronchitis at the bases. The nervous system was examined in detail. There was no optic neuritis, squint or nystagmus, and, in short, no evidence of any irritation or paralysis of any of the cranial nerves. The knee-jerks were normal, the plantar reflexes normal, and no evidence of any disease of the nervous system could be found, with the exception of a slight positive Kernig's sign in each side. A lumbar puncture was performed. The cerebro-spinal fluid was found to be purulent.

Professor Walker Hall's report on this was as follows:—"Cells: polymorphonuclear 80 per cent., endothelials 15 per cent., lymphocytes 5 per cent. Culture: gram negative, intracellular diplococci (meningococci) found."

The patient was given urotropin gr. x every six hours, and 30 c.c. of Flexner's serum was injected intra-spinally, after a similar amount of cerebro-spinal fluid had been removed. The patient, however, became more deeply unconscious, and died on the morning of the 18th, *i.e.* the day after admission, without the occurrence of any further abnormal sign in his nervous system.

Case 3.—E. G., aged 36, female, wife of Case 2, was admitted to the Bristol Royal Infirmary on April 28th, 1913, with pain

in her head. A week before admission she woke up with an attack of shivering, followed by vomiting. During the day she became seized with acute generalised headache. The headache, vomiting and shivering continued for four days, when she noticed sharp, stabbing pains in her back, which lasted about a minute at a time. These pains had disappeared on admission. On admission her face was covered with perspiration. Temp. 103; pulse 100; resp. 26. There was some stiffness in the muscles of the neck; the knee-jerks were slightly exaggerated, but there was no other sign of any abnormality in the nervous system. The fundi were perfectly normal, and there was no trace of any Kernig's sign. She could answer questions perfectly rationally, and gave a history of old chorea and of previous stiff necks, and stated that she was quite sure her neck on previous occasions had been as stiff as it was then. The heart was slightly enlarged, and at the apex a sharp first sound was heard, preceded by a presystolic murmur. Nothing abnormal was found in the lungs or abdomen. The diagnosis appeared to be between a rheumatic stiff neck and possibly cerebro-spinal meningitis. It was decided to administer salicylates, and if necessary perform a lumbar puncture. She was first given aspirin and then large doses of sodium salicylate. The temperature fell from 104 to 97.2, but there was no improvement in her condition, and on April 30th the temperature rose again. A lumbar puncture was therefore performed, when a quantity of milky fluid was obtained, in which the meningococcus was found by Professor Walker Hall, who also reported that there was no phagocytosis by leucocytes. The neck remained stiff, Kernig's sign remained absent, the fundi remained normal, and no fresh nervous sign developed. She was then put on urotropin, and 30 c.c. of Flexner's serum were injected intra-spinally. On May 1st there was a very slight blurring of the right disc, and an indefinite Kernig's sign appeared, tache cérébrale also being evident. The patient was rather drowsy, breathing was irregular and associated with a good deal of yawning; vomiting occurred five times. On May 2nd 30 c.c. of cerebro-spinal fluid were withdrawn, which was less turbid than before, and 30 c.c. of Flexner's serum were injected. She was still more drowsy, and the right disc was a little more blurred. Temp. 102.6; pulse 90; resp. 40. Other nervous signs as before. On May 7th improvement had continued, but she was still rather drowsy. The temperature for two days had not exceeded 99.4.

NOTE.—May 19. Progress has been uninterrupted. She now feels quite well, and has no abnormal physical sign in her nervous system.

ON THE OCCURRENCE OF SEROUS PLEURISY DUE TO PNEUMOCOCCI AND TUBERCLE BACILLI.

BY

F. H. EDGEWORTH, M.D. Cantab., D.Sc. Lond.,

Physician to the Bristol Royal Infirmary and Professor of Medicine, University of Bristol.

A. B., male, aged 32, was admitted as an in-patient to the Bristol Royal Infirmary on May 2nd, 1910, complaining of pain in the left side of the chest. He was found to have a temperature of 101° F., a pulse-rate of 84, and a respiration rate of 34 per minute, and to be coughing up a large quantity of mucoid fluid. On further examination, it was discovered that there was a left-sided pleuritic effusion, so extensive as to displace the heart to the right side, and to make the spleen palpable below the left costal margin. The blood contained 9,860 white cells per c.m., and a differential count showed 73 per cent. polymorphonuclears, 15 per cent. small lymphocytes, 10 per cent. large lymphocytes, 1 per cent. eosinophils, and 1 per cent. basophils. The temperature was found to persist, and the effusion to show no signs of lessening, so on May 4th he was tapped, and 24 oz. of a clear yellow serous fluid withdrawn from the left side of the chest.

This pleuritic fluid contained pneumococcus and staphylococcus albus, the latter probably due to skin contamination. Cytodiagnosis of the pleuritic fluid showed 95 per cent. small lymphocytes, 3 per cent. endothelials, and 2 per cent. polynuclears. Pneumococci were also present in the expectorated mucoid fluid.

Now pleural effusions caused by pneumococci are generally purulent, rarely serous in character. The singularity of the case was heightened by the fact that the cell-count of the pleuritic fluid showed 95 per cent. small lymphocytes. For it

has been stated by Widal and Ravaut that " when the dominant cells present in a pleural effusion are small lymphocytes, the probable cause of the effusion is the tubercle bacillus ; when the dominant cells are the polymorphonuclear leucocytes, the probable cause is some pyogenic organism."

It seemed advisable, therefore, to make further investigations. A guinea-pig was inoculated with some of the pleuritic effusion. The report returned was: " The experimental animal, weighing 180 grams, died on June 25th, six weeks after inoculation, having gained 90 grams in weight. Fourteen days before death there was a small local lesion. P.M.—There was a generalised tuberculous infection with tubercle bacilli present in the lesions."

It thus became evident that the serous pleurisy was due to a double infection—by pneumococci and tubercle bacilli, whilst the bronchial secretion was due to pneumococci.

Such a causation of a serous pleural effusion is somewhat exceptional. Thus Gee and Horder (in Allbutt's *System*) state that 16 per cent. of purulent pleuritic effusions are due to multiple infections, but they do not mention the occurrence of multiple infections in the case of serous effusions, which are generally due either to tubercle bacilli, or to pneumococci or to streptococci.

The further history of the case may be briefly recorded. The pleuritic effusion rapidly re-accumulated, necessitating a second aspiration on May 10th, when 96 oz. were removed. After that date slow absorption of the remaining fluid took place. The bronchial secretion and the hectic temperature gradually subsided in the course of four weeks, and the patient left the Infirmary on June 25th apparently quite well. He was advised to change his occupation—of a house painter—to one of work on a farm or garden.

WHAT IS AN ACCIDENT ?

BY

CHARLES CORFIELD, of the Middle Temple, Barrister-at-Law, M.R.C.S., L.R.C.P.

THE consideration of accidents is a matter which comes to most of us sometime or other in our professional work, mainly, of course, arising out of the Workmen's Compensation Act, but sometimes under common law as well ; and therefore a review of those decisions which have helped to define what an accident is may not be out of place at a medical meeting.

It is curious that neither of the Workmen's Compensation Acts contained this definition, so that the meaning of the term has tended to vary case by case. Prior to these Acts little difficulty had ever arisen in common law cases of negligence, as running-down accidents or railway accidents, and as regards workmen before these Acts negligence was the only claim for accident that could be sustained by a servant against a master. But these statutory enactments inaugurated a new principle in English law, and so gave a legal subtlety to the word "accident" that it never had before. The first Act was limited in scope, but the 1906 Act was of general application, and also made certain diseases statutory accidents as well. The first definition of an accident that I am able to find is in Stephen's *Commentaries*. He says in effect an accident is any mishap or untoward event which is not expected or designed, whether it be caused by the negligence of another person or not. This definition falls very short of the modern interpretation of accident. Many occurrences which cause injury are not unexpected, but a highly probable act of the workman himself. As the late Lord MacNaughten once said, a man may injure himself by doing some stupid thing and it is called an accident. The definition of Sir James Stephen, then, as it excludes the

consequences of carelessness from the meaning of the word accident, fails at the very outset as a guide to the interpretation of the term.

Another definition might be taken from a class of case where previous to the Workmen's Compensation Act the word accident was sometimes likely to come under review, namely in insurance and marine cases. Thus we find Mr. Justice Willes in 1868 holding that an exception in a charter party against riots and strikes or *any other accident* does not include a snowstorm. He says an accident is not an ordinary occurrence, but something which happens out of the ordinary course of things. A snowstorm, however, is one of the ordinary operations of Nature, and may be described rather as an *incident* than an accident.

Yet in the comparatively early days of the Workmen's Compensation Act we find that the effects of frost were held to constitute an accident, and later on the death of a workman by lightning was similarly held to be an accident too. So as judgment succeeded judgment the meaning and scope of the word became amplified and extended.

Again, in the well-known case of Fenton *v.* Thorley, it was laid down that to be an accident within the meaning of the Act there must be something fortuitous or unexpected in the occurrence. Fenton was a man in the employ of Messrs. Thorley. Whilst at work one day upon a machine he felt something which in giving evidence he described as a tear in his inside, and it was found that he had a rupture. In the medical evidence it was said that the hernia was a physiological one, which I suppose meant a congenital condition. In other words, that it had existed before, but that a severe strain had brought it down. Here we have an extension of the word accident to a condition which existed as a concealed defect, and which another workman without this congenital weakness doing the same work would not have contracted; and this is the first important departure from the word accident in its restricted sense.

The next case of importance which enlarged the scope of this much-debated word was Brinton *v.* Turvey. A wool sorter

contracted anthrax. (This, I may add, was before it was scheduled as an industrial disease.) In the course of the case on appeal, Lord Robertson said: "Anthrax is a disease, and unless the contraction of an infectious disease is an accident in the sense of the Act, I do not see how this judgment can stand. If it does stand, then in every case in which a man dies of any infectious disease contracted in the course of his employment all he has got to do is to get a doctor to say that a micro-organism did it, and the accident is there." The majority of the "House" was against Lord Robertson; so an infectious disease was by a legal decision from the highest tribunal included in the category of possible accidents under the Workmen's Compensation Act.

We now reach a further process of development in the meaning of the much-suffering word, for we come to a class of case where disease has been produced by a continual process rather than by a definite event, and yet the result has been held to be an accident.

Thus in Ismay & Co. *v.* Williamson a workman was employed as a trimmer on board the s.s. *Majestic*. He was a man of feeble physique and health. His work lay in the stokehold, where the heat was great. He developed on the second day symptoms of heat stroke and dropped in a faint. He was carried to the ship's hospital, and became violent and died from exhaustion two hours after leaving the stokehold. The condition of the stokehold was normal and ventilation good, and other stokers had worked for years in such conditions without any serious effects.

By a majority the House of Lords decided that this was death arising from accident. In the course of his judgment, the Lord Chancellor said what killed the man was a heat stroke coming suddenly and unexpectedly upon him while he was at work. Such a stroke is an unusual effect of a known cause, often no doubt threatened, but generally averted by precautions which experience in this instance had not taught. It was an unlooked-for mishap in the course of his employment. The judgment of Lord MacNaughten on this occasion is interesting, though he was in the minority. He said: "It does not seem to me to come under the description of a personal injury by

accident. The death was due to the physical state of the workman more than the nature of his employment."

Now, side by side with these two cases I have mentioned there are two other cases also of considerable interest, viz. Brodrick *v.* London County Council and Eke *v.* Hart Dyke (1910). In the first case the workman, according to the medical evidence, contracted enteritis from inhaling sewer gas for a period of six hours, and the result of the enteritis was to accelerate existing heart disease, and so precipitate the incapacity from heart trouble which sooner or later would have overtaken him. This was held by the Court of Appeal not to be a case of accident.

The case of Eke *v.* Hart Dyke, which I think is one of the last leading cases on this subject, still further confirms this decision I have just mentioned. It was a case in which the caretaker of an empty house was told to lay open the drains, manholes and cesspools for inspection. The man did this on several occasions, and becoming ill, died in the following October from poisoning contracted from the drains. It was held that this was not an accident. The Master of the Rolls, in giving judgment, referred to the anthrax case, and pointed out that in that instance the anthrax was due to the circumstance that at a particular time and at a particular place the infection came into the man's eye, and his lordship appeared to consider that that constituted a satisfactory ground for distinction. But if a particular event happens at a particular time and at a particular place, what about Ismay's, the stoker's case, where the man had worked two turns already that day in the stokehold and part of a third turn. Was there in that case any particular time at which the occurrence of a given event could be definitely ascertained and distinguished as a separate occurrence ? His collapse no doubt took place at a definite time, but his collapse was the result of an accident, and not the accident itself, because it was, so to speak, presumably the cumulative effect of several hours' excessive heat. If the test of a definite event at a definite time has to be applied, what length of time is sufficient for the purpose ? Must it be an

instantaneous effect, or may it be a slowly-operating process covering a period of time ? If so, how long a period ? In Ismay's case the man had been working for several hours on the second day, and there is nothing such as ventilation block which fixes any definite event during these periods. One naturally asks why is the six hours during which Brodrick worked in the sewer too indefinite, while the longer period during which Ismay's worked is definite enough.

These cases I have above illustrated are sufficient to show how difficult the question of accident is. The Master of the Rolls himself says the words injury by accident have undoubtedly created great difficulty. "I feel," said a recent Lord Chancellor, "that in construing this Act of Parliament there is a risk of frustrating it by excess of subtlety, which I am anxious to avoid." These difficulties are not due to a conventional inflexibility of the judicial mind, but to a trait inherent to any words that define by limitation, namely that wherever the boundary-line be drawn some cases will always be found to fall extremely near on either side. The solution of this vexed question might be attempted in several ways. Some day an ingenious definition of the word accident might be evolved which will steer between these dangers I have outlined above ; then more importance might be attached, not to the quality of the blow, so to speak, but to the condition of the recipient. Something, in other words, of the principle of contributory negligence might be introduced, and it could be made a good defence to a claim to show that the workman had a concealed defect which contributed equally to the condition, such as an aneurysm, a congenital hernia, and so on.

These restrictions would, however, if pushed very far, tend, I am afraid, to defeat the purpose of the Acts themselves, and therefore would not be tolerated.

Another suggestion is that all employees should undergo a physical examination, which is already done in the case of railway and other big companies, and as they pay a fee, from the point of view of the doctor such a plan has much to recommend it. This, however, without the privilege of

contracting out, would swell very much the ranks of unemployment, and would spell greater hardships than no compensation at all.

Lastly, I think, perhaps, the most practical solution of the difficulty, in regard to workmen's compensation, would be to include accidents among the benefits of the present Insurance Act. The only important difference this would make would be that employers would pay their premiums to the State instead of to the Insurance Companies.

The latter, who always aver that Workmen's Compensation does not pay them, would no doubt be up in arms at this interference with their widespread philanthropy.

THE TUBERCULOSIS OFFICER AND HIS WORK.

BY

LATIMER J. SHORT, M.D., B.S. Lond., M.D. Brist., D.P.H. Cantab., Fell. Roy. Inst. Pub. Health,

County Tuberculosis Officer for Somerset; late Chief Tuberculosis Officer for Woolwich.

THE tuberculosis officer is a comparative novelty. His immediate action and eventual position in medicine are as yet uncertain, and if the following description of him is somewhat diagrammatic, the writer pleads these circumstances as his excuse.

Two years' experience in city and county has indicated that the value of the tuberculosis officer's work will be in proportion to the extent to which he fits into his special place on the field.

He resembles the scrum half-back, and has to co-operate with nearly everybody in existence, if he is to be a permanent success. This alone makes it impossible to lay down any rigid boundaries, or even to give a fixed form to his actual

administrative department; but the following details apply to a county which is just beginning to realise its immediate needs.

The county tuberculosis officer finds he has really to represent, and be able to supervise, the whole general scheme of tuberculosis work in his area. Prevention and treatment cannot, and certainly should not, be separated, and prolonged special and general clinical experience is not merely an essential but the *first* essential. Experience in treating tuberculosis in general private practice is one of the most important parts of a tuberculosis officer's education.

The coming of the National Insurance Act is merely a detail, and the tuberculosis officer must not let his work be cramped down to that of a mere insurance officer. The attack upon tuberculosis is a far more comprehensive movement, of which the Act supplies one particular only, and it was really in process of formation long before the Act was thought of.

Dealing first with the clinical side of his work, the tuberculosis officer has to advise the Insurance Committee as to the form of treatment which should be arranged for every patient who applies for "Sanatorium benefit." This misleading name includes all forms of benefit for all classes of tuberculous insured persons, even if it be a pair of blankets and a jar of malt extract. Obviously, he should meet the patient's own medical man when he has one, and talk over the whole matter with him, especially considering the patient's surroundings and facilities for home treatment. Unfortunately, in a scattered rural district it is often impossible for the tuberculosis officer to visit all the cases, or for the medical man to visit the central office, and correspondence has to replace the desired interview—a much less satisfactory method, especially when the tuberculosis officer has not yet received details of the patient's home conditions. In such cases I usually postpone the ordering of such extras as milk, bedding, an open-air shelter, etc., until more information is forthcoming.

Cases are also frequently referred to the tuberculosis officer by their medical attendant for help in diagnosis, either clinical

or bacteriological, or for special tests, and these must be carefully investigated and reported on. School children form a large proportion of such cases, and others are sent for suggestions as to special treatment, especially where hospitals are few and far away.

On the *preventive* side, the tuberculosis officer must to a large extent be in charge of the details of the tuberculosis public health work in the whole of his area, under the general guidance of the county medical officer of health, and must be in constant touch with the tuberculosis health visitors and with the district medical officers, whose work he can materially lighten on one side even if he adds to it on others.

He must also be the connecting-link between the various agencies—professional and lay—now at work on the same campaign, which are really complemental to each other, if brought properly together, instead of being a mutual annoyance as so often happens at present. The securing of some material relief, an extra bedroom, temporary employment, and similar help, is often the most important part of the whole treatment of a case of tuberculosis. It is obvious that in a scattered county area these principles cannot be followed out without the provision of a large number of local centres in various parts of the county, where the tuberculosis officer can meet the patient and his doctor, can receive any sent to him for advice, can meet lay helpers, and can store such sundries as thermometers, sputum flasks, charts, cod liver oil and malt.

The local health visitor must make the same place her centre, keeping all her notes on cases and papers there for the information of the tuberculosis officer, to whom she reports weekly. It is convenient to call these " Dispensaries," although not much dispensing is done there, and they are the essential feature of a county scheme. There are already six in Somerset, and others are quickly to follow. They are all visited regularly at the same day and hour each week. It is at these " clinics " that cases for sanatorium beds are selected from among the many that one would like to get away from their homes. If a patient cannot come to the dispensary he is, as a general rule,

unsuitable for a sanatorium at the time, but there is still grave difference of opinion among the profession as to what cases should and should not be sent to sanatoria.

The greater ease of getting patients away, owing to the Insurance Act, has resulted in persons being hurried off who are entirely incapable of benefiting themselves by the special treatment, and are merely a source of danger and disturbance to the really good cases at the sanatorium. A continuance of this may make the benefit an evil, and bring discredit upon the whole sanatorium scheme. If it is necessary, however, that the advanced and incurable should not be mixed with early cases, it is equally necessary that they should not be left to infect their own relatives at home, and the county proposes to establish camps of portable and cheap but cosy wooden buildings, preferably grouped round some small permanent building, for those in this condition. Some patients will not require to stay a great length of time, and this will redeem the place from the stigma of a "death hole"; but others will require treatment here for months or years until their death, and the provision of every extra comfort that can be obtained must make up for their compulsion.

The tuberculosis officer will soon have to face the question of tuberculous children, excluded from school for months or years, and steadily going to the dogs, body and soul, in sunless and airless courts and houses, with nothing to do, and no energy to do it. In an urban area, an open-air class or school is a cheap and eminently successful remedy. Anæmic and so-called "pre-tuberculous" children can be admitted and taught separately. It is only a partial provision, and in a scattered area it is obviously impossible, as these children cannot travel far, and a residential open-air school is the only thing. This should be centrally situated, and combine nursing and teaching, with a resident medical officer in charge. The tuberculosis officer should help to select cases for the school, many being originally referred to him by the school medical inspectors.

Experience has shown that children do not profit by

institutional treatment, unless it is over six months in duration, and the normal stay at the open-air school should be one year, renewable if necessary. The actively tuberculous, in the infective stage, would be accommodated in a separate block, and would not mix with the others, schooling being inadvisable for them for the time being.

Recent investigations all go to show that pulmonary tuberculosis is far commoner in children than has hitherto been supposed, that its type and course differ from that seen in adults, and that its tendency to spontaneous apparent cure is very great, so that most of the cases are discovered only when active search is made for them. One says "apparent" cure because it is tempting to think that the great incidence of phthisis in the young adult is mostly but a recurrence of a previous, often unnoticed, infection in childhood.

This ushers in the vexed question of "contacts," by which one means the near relatives of a known case who are living with him in the house. It has been shown by Halliday Sutherland and others that there is a much greater incidence of tuberculosis in these than in people who are not thus described, owing to their close association with the sufferer, and it is only by a systematic and thorough clinical examination of these that the tuberculosis officer or anyone else can hope to get hold of the very earliest cases, before they are bad enough to seek medical advice on their own initiative.

In Woolwich, during 1911–12, out of 842 "contacts" examined clinically, I found no less than 291 who were either affected or so suspicious that immediate treatment was desirable, while as many as 63.5 per cent. of our patients (nearly 1,000 in number) apparently caught their disease from a previous case in the family and house. The difficulty is—"By whom should they be examined?" Members of a family are frequently under different doctors, and one could not properly examine the patients—either present or past—of a colleague.

In London general practice I found that any attempt to examine contacts was resented and suspected by the patient

as an attempt to " make work," even if no fee were asked, and it was found better that an outside man should make the examination, and report to the practitioner attending the original case. After all, the only important thing is that the contacts should be thoroughly examined,and the tuberculosis officer is satisfied if he knows that each one can be accounted for, and that they are not being dropped between two or more stools.

Another very important branch of the work is the investigation and reform of the housing, sanitary, sleeping and economic arrangements, for bad housing means tuberculosis. This is women's work, and I have never found it really successfully accomplished by mere men, however tactful and earnest. It is perfectly true that many practitioners make a point of most carefully investigating these matters themselves, but it is also true that others cannot spare the time, or have not the inclination to do so, and this being so, it seems best for the health visitors to visit all cases—satisfactory and doubtful alike, if a really comprehensive scheme is to be followed. The tuberculosis officer does no visiting unless he receives a request from the medical attendant to do so, or in consultation with him, or on bedridden cases applying for " sanatorium benefit."

As to treatment by the tuberculosis officer, there will always be cases which cannot afford the long and continuous treatment that tuberculosis demands if success is to be attained, and for whom no other provision is made. These should be treated at the dispensaries, and their number will depend on what arrangements are made with the profession in the future for the further attack on this widespread disease. It would be a mistake to deprive the tuberculosis officer of all experience in treatment, apart from the question of expense, as he then gradually ceases to be a clinician, but co-operation with the general medical practitioner must be maintained—and should not be difficult to maintain—in this as in all other branches of the great scheme against tuberculosis.

The chief difficulty of the future will be to prevent the administrative side from swamping the clinical, for when the

tuberculosis officer develops into an office machine, however efficient, his usefulness to medical science will be in very serious danger of coming to an end.

THE THIRD SOUTH MIDLAND FIELD AMBULANCE.

BY

Lieut.-Colonel A. W. PRICHARD, V.D.,
Late Officer Commanding.

IN the autumn of 1907, while the Volunteers were still in existence and not in a very satisfactory condition as regards numbers and efficiency, the formation of the Territorial Force in their stead was one of the topics of the day. I was then Brigade-Surgeon of the Portland Volunteer Infantry Brigade, and hearing that the Director-General of Medical Services was about to address the medical men of Birmingham with reference to the raising of R.A.M.C. units in the grouped regimental district in which both Birmingham and Bristol are situated, I urged the claim of Bristol to take its share, and argued that with its excellent hospitals, medical societies, University College and Medical School, to say nothing of the keenness with which men had joined the Artillery, Engineer and Infantry Volunteers, it could not well be left out. It was therefore decided by the War Office that of the three field ambulances and two mounted brigade field ambulances that serve each division, one field ambulance should be allocated to Bristol, and I was asked to raise it and take command.

On January 11th, 1908, a public meeting, under Colonel Savile, C.B., and a meeting of medical men of Bristol and the neighbourhood, under Dr. Michell Clarke, were called, and at both of them Colonel Russell of the War Office was present, and explained the organisation of the medical services. A resolution was passed approving of the steps that had already been taken,

and I had the satisfaction of receiving the names of several officers who were willing to serve under me.

As at that meeting of the profession the real start was made, it may be not unseemly if I give details of the history of the Field Ambulance for the five years that I have had the pleasure and honour of being in command.

No real recruiting was allowed before April 1st, 1908, when the transfer of the Volunteers to the Territorial Force was to take place. I could only get promises of names. King Edward was to come on the 9th of July to open the docks at Avonmouth, and until I had eighty men—a third of my establishment—the unit could not be recognised, and could not be clothed by the County Association. With the assistance of Major Young, many meetings were held, and the work of organisation made good progress. Our efforts were ably supported by Dr. Rogers, Dr. Green and Mr. Lambert (quartermaster), who had expressed their desire to take commissions in the new Field Ambulance. Colonel Woodward, of the Bristol Rifles, kindly allowed us part of his premises as head-quarters, and drills, lectures and officers' rides were begun. The head master of the Grammar School let us drill on the Grammar School ground, and we got our eighty men just in time to clothe them and drill them enough to fall in and help line the streets on the occasion of the King's visit. That was the first public appearance of the Bristol Territorial R.A.M.C.

Recruits came in rapidly, and in August we went to Swanage to camp for a week as a medical corps attached to the Gloucester and Worcester Brigade. We were very new to the work, and there were difficulties to be overcome, the chief of these being with the heavy transport and the railways. The second week of that fortnight was at Perham Down, Salisbury Plain, where we were separated from the Gloucesters and brigaded with the 1st and 2nd Field Ambulances from Birmingham, whose acquaintance we then made for the first time. During this week we left camp early one day and did not return until six o'clock next evening, bivouacking at night in a field with many other troops on the side of the Avon above crossing " A." After

a long field-day and a march past on Knighton Down, we had a ten-mile march back to camp.

Our first camp over, after a rest for a month or so, we began lectures and drills. We started a band, and had a first church parade of our own, which General Raitt attended, with the result that recruits came in willingly, and by the end of the winter we had recruited up to full strength, both in officers and men, a result that I believe no other Field Ambulance in England that had not had a bearer company of the old Volunteers to start it had achieved. We have been above strength ever since.

On April 1st, 1909—our first birthday—my officers were Major Young and Lieutenants Rogers, Mather, Green, Lavington, Moxey, Coleridge, Corfield and Lambert. We were busy with lectures, company and stretcher drills in the Grammar School ground, bugle practice, and signalling, and in July had the satisfaction of receiving from the medical profession of Bristol, in recognition of our efforts, a drum-major's staff, the presentation being made at the Drill Hall by General Raitt. That summer the fortnight's camp was again at Swanage, and a very enjoyable camp it was. We went down nearly full strength with all transport complete. We had the best position in the Brigade Camp, on top of a hill where there was welcome shade of trees. At the bottom of the hill was a large, flat meadow, which served the purpose of parade ground, cricket ground, and riding school. Looked at from the point of view of a pleasurable outing, at a pleasant seaside place, with bathing parades, cricket and country route marches, the camp of 1909 was a great success.

After this camp the officers decided to run their own mess. A business arrangement was entered into among the officers, so that all tent equipment and table necessaries should belong to themselves. All this has to be taken to camp every year, and a mess president is appointed to engage a cook and waiters and be responsible for ordering the necessary provisions. We have found this plan to work very well.

Nothing of great importance occurred during the next

winter. All the officers worked well; they had to pass their examination for certificate B, and things went very smoothly, except as regards the difficulty of getting our own head-quarters. Next summer, 1910, on Salisbury Plain a very pleasing honour was conferred upon the Bristol unit during this camp. A complete Field Ambulance of regulars was in training at Tidworth, and were to be inspected by the Duke of Connaught. It so happened that upon the day of the royal inspection I had been invited by the commanding officer of the regulars to bring my territorial corps from Perham Down to watch the work of the regulars. The Bristol unit was halted some little distance away from the scene of the operations, and saw the Duke arrive with his staff. His Royal Highness had been upon the ground but a few minutes when he observed the Bristol men in the distance, and he at once sent an officer with a command that I should bring my men to the ground where he was holding his inspection. We were allowed to watch the full routine of the work properly done, and afterwards greatly honoured at being ordered to march past, our band playing the R.A.M.C. march.

In 1911 the officer commanding the Bristol Rifles found that it was not convenient to harbour us any longer, and we had to seek shelter elsewhere. We therefore engaged the Hannah More Rooms in Park Street, where there were rooms for lectures, quartermaster's stores, sergeants' mess, and a small hall for drill. Our head-quarters at Kingsdown were being built. We tried one evening parade on Brandon Hill, but it was impossible to carry it through with decorum, owing to the huge number of children that were attracted by the bugles. This year we went full strength to Towyn with the Warwick Brigade and the other Field Ambulances. It was lucky it was a dry fortnight, as our camp was on ground that wet weather would have turned into a bog. We had no rain until the day of our departure, and that was a memorable occasion, as owing to the coal strike no trains were running, and we thought we should have to stay longer than we liked. However, the men were allowed by the railway companies to come away on the afternoon of the day fixed, and it took twelve hours, nearly half of which was in a train with no

lights, to get home. The horses did not reach home until some days later. We came back to our new head-quarters, at the back of the "Montague," Kingsdown, which were nearly ready.

The chief event of the winter was the opening of the new head-quarters at Kingsdown, on November 25th, by Lord Beauchamp. The President of the County Association attended, accompanied by Sir Arthur Anstice, Colonel Savile, Captain Colchester Wemyss, and many of the officers of the territorial units. All who had been invited to attend made a tour of inspection, and expressed considerable satisfaction at the arrangements that had been carried out. Some old coach-houses had been re-roofed and turned into commodious wagon sheds for the ambulances, with a covered way for them down into the drill yard; another shed had been converted into a heavy storeroom, and a three-stalled stable had been fitted up as an excellent harness-room. A new lecture-room, sergeants' mess and large drill hall had been built, and the interior of Colston Fort House itself had been re-arranged for officers' room, orderly room, quartermaster's stores, and various offices. Lord Beauchamp performed the opening ceremony, and the function passed off very pleasantly. A fund was started to supply proper gymnastic appliances, and within two months we had a good gymnastic class at work.

The year 1912 will be remembered for two reasons—the week-end Whitsuntide camp and the summer drenching on Salisbury Plain. At Whitsuntide a week-end camp was held at Tockington, and four fine days were much enjoyed. The August camp was very different. It was situated at West Down South, a most exposed place and seven miles from anywhere. We marched in dry, and then had no day without rain. However, a great deal of real training was done. We were with the other two Ambulances, two infantry brigades, and many other troops. Much interest was taken in the flying from Larkhill, which took place whenever the weather made it possible; and the day which the men enjoyed most was a route march to Stonehenge and Amesbury. We took meat

and vegetables and stopped for two hours in the middle of the day, made fires and cooked it, and on the way back halted at Stonehenge and saw many aeroplanes flying close over us.

During this camp all field drill was by whistle and signal, no general word of command ; and a complete system of competitions for prizes in the different classes, such as company drill, stretcher and wagon drill, signalling, bugling, and the grooming and keep of harness and horses, was carried out.

I was greatly pleased at a sentence in the Special Sanitary Officer's (Southern command) report : " This is the unit with the best arrangements for sanitation in camp. A great deal of time and trouble is expended on it, and but for the deficiencies of the contractor it may be considered a model to others." The " deficiencies of the contractor " refers to the slowness of carting the refuse away.

At home during the last winter special attention has been paid to the social side of Territorial life. A great many 1908 and 1909 men signed on for four years' service, and to encourage some of them to re-engage as their time expired and to bring other friends in, a series of smoking concerts and a Christmas entertainment were held, with, I hope, a good result. My time for retirement came, and my last parade was a church parade at the Cathedral on February 23rd, and I took leave of the corps with many feelings of regret at a well-attended R.A.M.C. dinner on March 5th.

In conclusion, I wish to say that I do not take the credit of the success of the undertaking to myself. I have omitted in my remarks any reference to the N.C.O.'s, who have, many of them, given up a great deal of time after working at their various occupations all day to, first of all, getting their promotion by examination—and that examination no light one—and afterwards carrying out their duties as soldiers should. To them and to the whole of the officers the credit is due. Mess dinners (three or four in the year), officers' rides, officers' meetings, and camp have united us together like a happy family, and made the time to me very pleasant ; and I am sure the present commanding officer, Lieut.-Colonel James Young, will keep

up and enhance the prestige which I believe the 3rd South Midland Field Ambulance at present holds.

The following is a list of the officers up to April, 1913 :— Lieut.-Colonel A. W. Prichard, 1.4.08 ; Lieut.-Colonel J. Young, 1.4.08 ; Captain B. M. H. Rogers, 1.4.08 ; Captain J. S. Mather, 1.4.08 ; Captain T. Green, 1.4.08 ; Captain P. Moxey, 1.4.08 (transferred to the 6th Batt. Glo'ster Regiment for one year and then rejoined the Field Ambulance) ; Captain A. Coleridge, 23.3.09 (transferred to 4th Wessex R.A.) ; Captain C. C. Lavington, 15.1.09 ; Lieut. C. Corfield, 18.3.09 (attached to S.M.F.A.) ; Lieut. C. E. K. Herapath, 1.4.11 ; Lieut. G. Scott Williamson, 1.11.09 (transferred from York Mounted Brigade F.A.) ; Lieut. C. F. Walters, 18.1.11 (transferred from 2nd Southern General Hospital, retired) ; Lieut. and Quarter-master H. Lambert, 1.4.08 ; Chaplain Rev. Canon J. G. Alford, V.D., 1.1.10.

Progress of the Medical Sciences.

SURGERY.

The existence of **cervical ribs** has received more attention from anatomists than clinicians, but the possibility of such an abnormality should always be considered in obscure cases of vascular, muscular or nervous disturbance in the upper limbs. In a recent discussion on this subject at the Royal Society of Medicine, Dr. Farquhar Buzzard [1] said that for some years he had been in the habit of submitting to an X-ray examination almost all cases in which pain in the arms was a predominant symptom, and as a result of his observations he found that not only did a cervical rib cause pain, distributed longitudinally on either the radial or ulnar sides of the limb, but also might produce a typical acroparæsthesia with pain limited to the hand and fingers.

Speaking from the anatomical point of view, Dr. Wood Jones [2] thinks it certain that the vascular symptoms in the arm are not produced in all cases by compression of the artery, but

[1] *Proc. Roy. Soc. Med.*, 1912–13, v., Clin. Sect., p. 139.
[2] *Ibid.*, p. 95.

that just as pressure on the lowest cord of the brachial plexus produces the muscular and sensory changes, so it may in some cases also produce the vascular symptoms. It is the compression of the vasomotor fibres in the lower cord of the plexus which causes these symptoms. Mr. Percy Sargent[1] says in some instances the injury to the nerves is brought about by the bony part of a long rib, but in other cases the nerves are in close relationship with the non-bony part of the anomalous rib, which consists of a dense fibrous cord embedded in muscle, attached above to the rudimentary bone whilst below it is most commonly attached to the first dorsal rib. As the sternum rises in inspiration the posterior part of the first dorsal rib tends to be depressed, so that the band, the upper attachment of which is immovable, is tightened. In this manner the nerves are damaged, and the possibilities of such damage are increased by excessive use of the arm. A study of the skiagrams of these cases shows that the degree of the symptoms depends in no way upon the size of the bony portion, and probably the direction of the bony point of the rib is more important than its size.

The symptoms of cervical rib may be divided into motor, sensory and vasomotor systems respectively. Dr. Kinneir Wilson[2] classes the sensory symptoms as subjective and objective. In the former group we have tingling, numbness and " pins and needles," referred either to the ulnar or radial side of the hand more frequently than to all the fingers, and mostly unilateral. It is rare to find the pain starting from the region of the neck or shoulder, but generally in the forearm, hand or fingers, and most patients say that it always radiates in a downward direction. Objective sensory symptoms are variable. In some cases no definite alteration in cutaneous sensibility can be discovered ; in others there is a diminution or loss to all forms of cutaneous sensibility, the distribution of which is never exactly that of either the radial or ulnar nerves, and does not harmonise entirely with a root supply.

The motor phenomena are practically pathognomonic, and consist of two main types. The first of these, which may be designated the median type, is very frequent, and in it there is an early local wasting of the muscles of the thenar eminence, in which the muscles involved are the abductor pollicis and opponens pollicis alone, all the other thenar muscles, including the flexor brevis pollicis, being intact. The other type of muscular atrophy corresponds, roughly speaking, to an ulnar distribution ; in other words, we find general wasting of the interossei and an approximation to the *main en griffe*. In most of the cases of this kind it will be found that the patient complains of paræsthesia along the ulnar border of the hand and in the ulnar fingers, whereas in the cases offering the local thenar

[1] *Ibid.*, p. 117. [2] *Ibid.*, p. 133.

atrophy of the first type the paræsthesiæ are on the radial side. Occasionally muscular cramps in the hand and fingers are noted by the patient without any muscular atrophy being discoverable.

When vasomotor phenomena are present the limb may be smaller than on the opposite side, the hand may be pale or blue and cold, the radial pulse may be absent and gangrene sometimes supervenes.

In an analysis of twenty-nine cases, Mr. Percy Sargent[1] found twenty-six females and three males, with an average age of about thirty-eight years. The first symptom noticed by the patient was as follows:—Pain, seventeen; numbness, five; swelling or coldness of the hand, four; and clumsiness in the fingers, three. In nine instances the onset of symptoms was ascribed to the following causes:—Excessive sewing, two; use of a sewing machine, one; piano-playing, one; house painting, two; wearing a heavy overcoat, one; lifting a patient, one; and childbirth, one. In the last two the onset of symptoms was sudden. The symptoms for which the patient sought relief were:—Pain alone, four; pain and wasting, nine; pain and numbness, six; wasting and numbness, five; and wasting alone, five. In every case the abnormal ribs were bilateral, though rarely symmetrical; the symptoms, however, were bilateral in only five of the twenty-nine patients. As a rule, when the symptoms were unilateral, the arm on the side of the smaller rib was affected; thus in nineteen cases in which the relation was specially noted the symptoms were on the side of the larger rib in only eight.

Relief of the symptoms is obtained by removal of the abnormality, but the operation is not always easy. Sir Rickman J. Godlee[2] considers that most of the difficulties are avoided by making a transverse incision above the rib and not over it; whereas Mr. Thorburn[3] considers that the operation is most easily performed through a long incision straight down and well back on the neck, over the anterior border of the trapezius muscle. The latter had only met with cases in which there were combined vascular and nervous symptoms, and had not been called upon to operate for vascular symptoms alone, though he recognised that an operation might be necessary where there was marked congestion or anæmia or threatened gangrene of the part. As a result of twenty cases, Mr. Thorburn found that all were greatly improved. Pain was cured in four-fifths of the cases which could be traced afterwards; paralysis was cured in half the cases, though some weakness remained in the others; anæsthesia was cured in all, though recovery from this was slow, and the sense of coldness never entirely disappeared.

Operation is, unfortunately, often immediately followed by symptoms of more or less severe, though usually transient,

[1] *Loc. cit.* [2] *Ibid.*, p. 140. [3] *Ibid.*, p. 113.

character. Dr. Howell[1] was able to follow up twenty-five cases in which thirty operations—as in five cases bilateral ribs were removed—were performed by several surgeons. Of the twenty-five patients operated on, of whom two only were males, there were symptoms due to operation in eighteen instances. Such symptoms were pain, more or less severe, in neck, shoulder or arm, and muscular weakness, which varied considerably both in extent and duration. In eighteen cases pain was complained of, in some instances being very severe. The duration of this symptom was, as a rule, from one to three months; but in one case, in which there was a decidedly neurotic element, pain was still complained of seven years after operation. Fifteen patients showed motor symptoms as a result of operation. The usual complaint was of weakness of the whole arm, lasting from two weeks to three months, but in four cases the symptoms were more pronounced. In only twelve instances was there absolutely no motor or sensory disturbance following operation.

In dealing with the ultimate effects of operation, Dr. Howell divides the cases into four classes, according to the symptoms they exhibited:—1, vasomotor symptoms only, two cases; 2, subjective sensory symptoms only, five cases; 3, motor symptoms and subjective, but no objective sensory symptoms, eleven cases; 4, complete cases, which include all the above symptoms, and exhibit in addition objective sensory disturbances. He summarises the results of operative treatment of these cases as follows:—In a large proportion of cases some symptoms, such as pain and weakness in the arm, may be expected to follow the operation, but not to last more than about three months. The vasomotor symptoms, which are present in nearly all cases, will be certainly improved, and in the majority of cases pain will be relieved or cured. With regard to weakness and atrophy, the expectation is that the operation, if not delayed too long, will greatly improve this condition. There is not, as a rule, complete restoration of the wasted muscles nor complete recovery from the vasomotor disturbance.

JAMES SWAIN.

PATHOLOGY.

Perhaps the most signal advance made during the past year or so in bacteriology is Noguchi's success in securing a pure **culture of the treponema pallidum** in the test tube.[2] An extract made from such a pure culture has been used as an antigen in the Wassermann technique for the diagnosis of syphilis, and in preparing an extract for use on the lines of von Pirquet's reaction

[1] *Ibid.*, p. 127.

[2] Noguchi, *J. Exper. Med.*, 1912, xvi. 194.

in tuberculosis. Noguchi further announces the discovery of the treponema pallidum in the brain of a few cases of general paralysis of the insane.[1] Making use of the pallida antigen for comparative study with the original Wassermann antigen prepared from syphilitic liver and other tissues, Craig, Nicholls and Noguchi have demonstrated the superiority of the original antigen in securing a diagnosis in syphilis.[2] In the tertiary stages and after treatment the pallidum antigen appears to give more delicate indication of an actual immunity response. Noguchi claims that in the pallidum antigen we have a means of determining the defensive activity of the host, whereas the Wassermann antigen gives an indication of the infective virility of the active agent, the treponema pallidum.[3]

The value of the **intradermic Luetin reaction** in syphilis remains undetermined. The records[4] of the test indicate a decided specificity, curiously enough almost confined to the tertiary stage of the disease. It is significant that a very large percentage of cases treated with salvarsan and giving a negative Wassermann reaction gave a positive Luetin test.[5] Until the explanation of these findings are clear it would be as well not to claim that a negative Wassermann reaction after treatment indicates freedom from infection.[6] There is much that is obscure and little that is obvious in these diagnostic reactions. That they are all of one type seems undoubted from whatever source the protein employed is derived. There is considerable evidence to associate these reactions with the phenomena of anaphylaxis.[7] Anaphylaxis, according to the authorities, would appear to be a purely physical reaction, probably an absorptive function of a colloidal body.

The **tuberculin reactions** fall into this category. They have been the subject of much study. The general conclusions on the basis of considerable experimental data make it certain that there is little reliance to be placed upon them in clinical diagnosis.[8] It would almost seem that the tuberculin reaction is more an expression of some passive susceptibility rather than any indication of the response of the body to infection by the specific agent. It is certain that these reactions are not a function of the specific toxicity of the organism.[9] Zinsser's

[1] Noguchi and Moore, *Ibid.*, 1913, xvii. 232.
[2] Craig and Nichols, *Ibid.*, 1912, xvi. 336.
[3] Noguchi, *J. Am. M. Ass.*, 1912, lviii. 1163.
[4] Robinson, *J. Cutan. and Genito-Urin. Dis.*, 1912, xxx. 410.
[5] Kämmerer, *München. med. Wchnschr.*, 1912, lix. 1534.
[6] Nobl and Fluss, *Wien. klin. Wchnschr.*, 1912, xxvi. 476.
[7] Belin, *Compt. rend. Soc. de Biol.*, 1912, lxxii. 692.
[8] Wyschelescky, *Ztschr. f. Tuber.*, 1912, xix. 209.
[9] Deycke and Much, *München. Med. Wchnschr.*, 1913, lx. 119.

work on the endotoxins of the typhoid bacillus [1] indicates a separate entity for the fever-producing poisons into which category the tuberculins fall, and casts some doubt on the endotoxic theories associated with that and other organisms. The very meagre justification for the use of tuberculin as a therapeutic agent rests upon this endotoxic hypothesis. It is as well to emphasise this relation between the tuberculin reaction and the condition of anaphylaxis, and to confess our comparative ignorance of the meaning of these phenomena. That anaphylaxis is a dangerous condition is well recognised. A recent observation of Bacmeister's [2] serves to emphasise this point of view. He has demonstrated that virulent tubercle bacilli appear in the blood stream of tuberculous persons after receiving an injection of tuberculin. Blood from four out of fifteen cases served to infect guinea-pigs with acute tuberculosis.

In view of the unreliability of the tuberculin reaction and our ignorance on the subject generally, it would be as well to abandon the use of tuberculin as a diagnostic, and little, if anything, can be said in favour of its use as a therapeutic agent. Its use as a prophylactic against tuberculosis in cattle would seem to be a menace rather than a protection. [3] Griffiths, [4] Weber and others indicate the possibility of actually creating tubercle bacilli carriers as the direct result of prophylactic inoculations of calves. All our experimental findings serve to indicate some extravagance in the claims of the tuberculin advocates. No doubt the neglect of tuberculosis as a subject for research has been in great part due to the satisfaction expressed by those who regard tuberculin as a panacea for the tuberculous.

In a recent issue of the *Journal of Experimental Medicine* Flexner [5] brings forward some further statistics on the use of **anti-meningococcal serum in the treatment of epidemic cerebro-spinal fever.** He has collected some 1,300 cases so treated. The results substantiate the earlier reports, and seem to prove conclusively that the serum has a very direct influence on the mortality rate. A reduction of some 20 per cent. must be looked upon with considerable satisfaction. The report deserves very careful study, especially with regard to the effect of the serum in protecting the patients from the rather disastrous sequelæ of the disease. The importance of early diagnosis is

[1] Zinsser, *J. Exper. Med.*, 1913, xvii. 117.

[2] Bacmeister, *München. med. Wchnschr.*, 1913, lx. 343. Laird, *J. Med. Research*, 1912, xxvii. 163. Hammer, *München. med. Wchnschr.*, 1912, lix. 1750.

[3] Weber, *Tuberculose Arbeiten*, 1910.

[4] Griffith, *J. Path. and Bacteriol.*, 1913, xvii. 323.

[5] Flexner, *J. Exper. Med.*, 1913, xvii. 577.

very strikingly emphasised by a table showing the effect upon the mortality rate of early injections of a serum.

Injected on	No. of cases.	Per cent. died.
First to third day	199	18.1
Fourth to seventh day ..	346	27.2
Later than seventh day ..	666	36.5

The earliest diagnosis can only be made from an examination of the cerebro-spinal fluid procured by lumbar puncture. Lumbar puncture is a safe and easy procedure, and ought to be resorted to without hesitation in any obscure meningeal condition. Another feature of these results is that they are the result of the employment of inoculation of the anti-serum by dural puncture; other methods do not yield anything like such good results. Probably it would be as well to turn the disease over to the surgeon for treatment as a septic fever.

As another outcome of the splendid research work of Flexner, **the epidemiology of epidemic poliomyelitis** has been still further elucidated.[1] Both in human and in experimental poliomyelitis the virus has been repeatedly demonstrated in the tonsils, the secretions of the naso-pharyngeal mucosa, and the discharges from the intestine.[2] The marked viability of the virus under adverse conditions, such as drying, low temperature, etc.,[3] and the fact that the virus remains alive in dust,[4] must be considered as making for a fairly complete explanation of the local spread of the disease. Of still further importance is the fact that the virus has been recovered from the naso-pharynx of persons in good health who have been in attendance or contact with acute cases. A very important point to be remembered is that the abortive and cured case can remain a carrier of the virus.[5] It still remains necessary to account for the spread over considerable areas and apart from the possibility of contact infection. Howard and Clark,[6] as the result of their research work, assign some considerable importance to the fact that the domestic fly can carry the virus on its body and in its alimentary tract. This in all probability affords an explanation of the marked seasonal incidence of the

[1] Flexner, *J. Am. M. Ass.*, 1912, lix. 273.
[2] Kling Wernstedt, *Ztschr. f. Immunitätsforsch.*, 1912, xii. 316.
[3] Levaditi and Danulesco, *Compt. rend. Soc. de Biol.*, 1912, lxxii. 543.
[4] Neustädter, *Deutsche Med. Wchnschr.*, 1912, xxxviii. 693.
[5] Landsteiner and Levaditi, *Compt. rend. Acad. d. Sc.*, 1911, clii. 1701.
[6] Howard and Clark, *J. Exper. Med.*, 1912, xvi. 850.

disease. It has also been shown that the blood-sucking insects, the bed bug, is capable of taking the virus with the blood from infected animals and maintaining it in a living state for seven days. This last observation has probably little if anything to do with the spread of the infection in man.

The elaborate researches of Lewis Carrol on **the cultivation of tissue cells in vitro** has started a new line of investigation in cancer research. With cells in more or less pure culture it becomes possible to determine the nature of the stimulus required to initiate mitosis and proliferation. Much interesting work has already been accomplished along these lines with tissue cells corroborating the findings of zoologists experimenting with the ova of fish, etc. The general results seem to indicate that physico-chemical processes depending more or less on the colloidal nature of protein play a considerable part in the process. It does not necessarily follow that the tissues themselves provide the stimulus, so that the possibility of some quite extraneous agent is not excluded. Apart from the question of the etiology of cancerous growth, this work will have immense value in physiological research generally.

G. SCOTT WILLIAMSON.

Reviews of Books.

Glycosuria and Allied Conditions. By P. J. CAMMIDGE, M.D. Pp. vii. 467. London: Edward Arnold. 1913. 16s. net.

The senior student and practitioner who is interested in diabetes and allied conditions will find a large amount of information in Dr. Cammidge's book, which we can thoroughly recommend as a painstaking and interesting monograph on this very complicated subject. Naturally a large portion of the work is taken up with questions of physiological chemistry and metabolism, but there is also a good deal of clinical description and not a few detailed cases, illustrating points brought forward by the author.

The amount of space devoted to the purely chemical and physiological chapters, though adding to the completeness of the author's treatment of the subject, might, we think, have been considerably reduced. The greater part of the material can be easily found in other books, and the space gained by this compression could well have been utilised in expanding the chapters devoted to diabetes and glycosuria, which suffer in places from a too brief and condensed method of treatment. The chapter on experimental glycosuria is a very adequate one,

and in some ways superior to those which follow. It may be read with great advantage by students who are going up for the higher examinations in medicine. In the chapter on transitory glycosuria, there seems to be a little confusion as to the headings of sections, which somewhat obscures the arrangement of the matter. The clinical distinction, however, between purely transitory glycosuria and those cases in which the symptom returns whenever special strain is put on the sugar metabolism is well brought out, and illustrated by some interesting cases. In the next chapter the account of the changes in the urine and blood in "true diabetes" is very fully given, but the symptomatology and complications are described in a rather perfunctory manner, and the author is tempted, as in the case of acetonuria, to digress somewhat considerably from the clinical aspect of the condition. The next chapter, on the pathology and diagnosis, suffers somewhat from condensation. Moreover, the author has not been able to avoid a partial repetition of some matters discussed in the chapter on experimental glycosuria. The diagnosis deals very largely with the question of pancreatic involvement and the various methods for determining the functional efficiency and organic soundness of the pancreas, on which the author is an authority. The chapter on metabolism begins with an account of normal metabolism, which is perhaps rather a luxury than a necessity in a work of this sort. The special metabolic anomalies of diabetes are rather briefly dealt with, but the matter on the whole is stated as clearly as its difficult character will allow. The ninth chapter, that on treatment, contains a number of valuable tables and charts which should prove of considerable practical use to those who wish to undertake the scientific as opposed to the haphazard treatment of their cases. It emphasises the need of individual tratment, and outlines a system based on a determination of the clinical data of the case. It is well worthy of careful consideration by all practitioners who can obtain these data and act upon them, though it is characterised rather by scientific precision than by clinical insight. The remaining chapters deal with subjects of somewhat academic interest, which are presented in an adequate manner, but not more fully perhaps than is usual in the larger "systems" of medicine. There are useful bibliographies at the end of each chapter and an index of nine pages.

The work as a whole is to be commended as a useful *rêsumé* of the present position of our knowledge and also as an interesting account of the author's special experience from the chemical standpoint. We hope that when a new edition is required more care will be taken in revising the proofs, and that the author will definitely make up his mind on certain points in orthography. Not only is he in doubt as to the final "e" in such words

as "glycerine" and "formaldehyde," but we also find "sterio isomer," "mutarotation," "anærobic" and "guiacol." Phlorhizin throughout is spelt "phloridzin," which seems a pity since attention has recently been called to this common error. Fractions of a gram are frequently coupled with a plural substantive, and there are numerous misprints of a more or less obvious character. These, however, are minor blemishes, and we congratulate Dr. Cammidge on the result of his work and the publisher on a very useful and well-set-up volume.

Cæsarean Section in Great Britain and Ireland. By AMAND ROUTH, M.D., F.R.C.P. Pp. 233. London: Sherratt and Hughes. 1911. 5s. net.

Dr. Routh's work contains tables of 1,282 cases of Cæsarean section by over one hundred living obstetricians and gynæcologists in Great Britain and Ireland, and is one of more than ordinary usefulness. It presents the views of obstetricians of all countries and schools on the indications and technique of this operation, and in the summaries at the end of the sections gives the general consensus of opinion on the more doubtful points.

The various indications for the operation are considered in detail. Naturally, the one which takes up most space is obstructed labour from contracted pelvis. The place of Cæsarean section in the treatment of this condition is most fully discussed. Comparison is made with the various alternative methods of treatment—induction of labour, embryotomy, pubiotomy; the difficult question of infected and "suspect" cases is throughly gone into, and the relative merits of the different methods of operating clearly set forth. The ordinary transperitoneal operation is the one most generally approved in this country; infected cases which need Cæsarean section, where an extra-peritoneal method would be most desirable, being best dealt with by Cæsarean section followed by hysterectomy.

The prevailing opinion on sterilisation as an accompaniment of Cæsarean section appears to be that the decision as to its adoption should be left, after the position has been clearly explained, to the patient, the favourite method of effecting it being by removal of the Fallopian tubes in whole or in part. A useful summary of conclusions brings this part of the subject to a close.

In the following sections the circumstances in which Cæsarean section may be called for in cases of uterine or pelvic tumours, uterine hemorrhage or constitutional crisis are considered, and an account is given of the history and present position of the operation of vaginal Cæsarean section.

The paper concludes with a series of mortality tables, showing the gradual diminution in the death-rate during the last decade, its variation according to the condition for which the operation is done and the type of procedure adopted, and the enormous influence on it of previous infection of the genital tract.

Appended is a very careful summary of the 1282 cases on which these observations are founded. The work is a complete account of the operation of Cæsarean section, and there is no point in connection with it on which ample material for forming an opinion and assistance thereto may not be obtained.

Flatulence and Shock. By F. G. CROOKSHANK, M.D. Lond., M.R.C.P. Pp. 47. London: H. K. Lewis. 1912. Price 2s.—These two essays are of much interest, and should be carefully perused. Common things such as "windy spasms" demand more consideration than they often receive. Among many other causes the author emphasises the importance of gas secretion from the walls of the stomach and bowels, to which air gulping is added to overcome the spasm that prevents eructation. The author concludes his essay with a practical suggestion likely to be of service not unfrequently: "Should any of you, from overwork, overmuch tea, overmuch tobacco, and what not, get a spell of neurotic flatulence and eructation with air gulping, nothing will give you so much relief as a bottle of the very best Crème de Menthe, sipped slowly and steadily until you are better." The author emphasises the importance of recognition of "delayed shock," when death may occur in an aged person a week or two after a slight fall that has involved fracture of the femur. Death is clearly the result of the accident, even although the obvious lesion appears to be of little danger to life.

Treatment after Operation. By WILLIAM TURNER, M.S., F.R.C.S., and E. ROCK CARLING, B.S., F.R.C.S. Pp. x, 247. London: University of London Press. 1912. 10s. 6d. net.—This small and most useful book describes in a simple way all the essentials of treatment after surgical operations. The opening chapters deal with the sick room, anæsthesia, asepsis, hæmorrhage, and operations on various tissues; and the rest of the book is concerned with the operations of regional surgery, ending with a chapter on the eye by L. V. Cargill. No attempt is made to review or discuss all possible methods of treatment, but only those which are in practical use by the authors are described. It is not, therefore, an exposition of anything new, but rather a helpful guide for students or house surgeons.

Gonococcal Infections. By Major C. E. POLLOCK, R.A.M.C. and Major L. W. HARRISON, R.A.M.C. Pp. xxi, 222. London:

Oxford Medical Publications. 1912.—The account given of gonorrhœa and its complications and of the other gonococcal infections given in this book is an excellent one. It is short, succinct and practical, and will be sufficient for the great majority of practitioners. Pathology, vaccine and serum treatment, and complications, both in the male and female, receive due attention. The most modern methods of treatment are described with sufficient detail to enable the practitioner to adopt them, and guidance is given in the selection of methods, a guidance based upon the writers' large experience. We can cordially recommend the book.

A Text-Book of Pathology. By J. GEORGE ADAMI, M.A., M.D., F.R.S., and JOHN MCCRAE, M.D., M.R.C.P. Pp. x, 17–759. London: Macmillan & Co. Ltd. 1912. 25s. net.—The book is admirably adapted to the requirements of the medical student, for whom it has been written. It is divided into two parts, dealing with general and special and systemic pathology. The authors have endeavoured to make clear and intelligible what is known concerning the deeper meaning of morbid states, and in so doing have certainly succeeded in giving a remarkably clear account of many abstruse subjects. The book is well illustrated by numerous engravings in the text, and also by eleven coloured plates.

Sleeping Sickness. By Captain F. P. MACKIE, I.M.S. Pp. 38. Calcutta: Superintendent Government Printing. 1912.—The pamphlet gives a summary of the work done by the Sleeping Sickness Commission (1908–1910), of which Capt. Mackie was a member. The Commission concerned itself chiefly with the question of sanitation and transmission of sleeping sickness by biting insects. Treatment, even if experimentally a success, is practically out of the question under the conditions prevailing in Africa. The reviewer ventures to consider that one of the most important results of this expedition is the observation that Glossina Palpalis fed on natives who have been treated with arsenic compounds and other drugs is capable of transmitting sleeping sickness (p. 27). Recurrent trypanosomes, after arsenic treatment, are often very virulent, and this fact emphasises the great danger which is involved in the treatment of thousands of natives with drugs which only cause a temporary disappearance of the trypanosomes. Such a mistake has been made by Koch, and is greatly to be regretted.

Microbes and Toxins. By D. ETIENNE BURNET. With a Preface by ELIE METCHNIKOFF. Translated by Dr. CHARLES BROQUET and W. M. SCOTT, M.D. Pp. xvi, 316. London: Wm. Heinemann. 1912. Price 5s.—**Clinical Immunity and Sero-Diagnosis.** By A. WOLFF-EISNER, M.D. Translated by

RAY W. MATSON, M.D. Pp. xiv., 184. London: Baillière, Tindall & Cox. 1911. Price 7s. 6d.—Both of these small books may be read with advantage by the student of bacteriology. Of the two, however, Dr. Burnet's book will be found to be more suitable for those not engaged in active bacteriological research. While it is not by any means a text-book on the subject, as its name might imply, it nevertheless deals with the broad underlying facts of bacteriology, at least so far as we know them. It begins with a rapid bird's-eye view of organisms in general, and their place in Nature, and gradually leads on to the great problems of immunity. As one would expect, the "phagocyte" dominates this part of the work, but the writer is broad enough to state fairly the humoralistic theory. Vaccines and sero-therapy are also discussed. It has been exceptionally well translated by Drs. Scott and Broquet, and contains much that is stimulating and suggestive. It can be read easily by the practitioner whose knowledge of the subject is limited, and should certainly be read by every student. The second book, while essentially one for more advanced workers in bacteriology, is too full of "Wolff-Eisner." We should not like to place it in the hands of a "tyro," if we desire him to have a fair idea of the subject. Not that it is of little interest, for it is full of suggestions, but it is too one-sided. The author is possessed of the new idea of anaphylaxis, and he attempts to make the observed facts of infection and immunity fit this idea. There is much in the book that is open to discussion, for much of it is entirely without proof. For the advanced researcher it is to be commended. For others, however, the first-named book will be of greater value.

Bacteria. By Dr. MAX SCHOTTELIUS. Translated by Staff-Surgeon HERBERT GEOGHEGAN, R.N. Second Edition. Pp. xii, 324. Ten plates. London: Oxford Medical Publications. 1912. 8s. 6d. net.—This is a popular, discursive, attractively-illustrated sketch of bacteriology for the general public. It begins with a survey of the position and use of bacteria in Nature; then describes bacteriological research methods, infectious diseases in general, and the prevention of infection by means of disinfectants, immunisation and other means. Next special diseases are taken as examples, and the work finishes with a chapter on protozoa. The advantages of public health administration and a knowledge of hygiene are indicated in the descriptions of small-pox and dysentery. Digressions are common. For instance, "Teetotal fanatics will not reach heaven" (page 7), and the opinion that teachers should rank "above the confectioners with their gay little tarts" (page 10); but surely the emphasis of the specialisation of bacteria, particularly the parasitic species dangerous to man, would have been more in place. The spoiling of fine furs (page 132)

is a serious matter, but the use of formalin in foods is a more serious objection, though this has escaped the notice of the author. These are, however, details. The book is easily read, and cannot obviously give very detailed information.

Puerperal Infection. By ARNOLD W. LEA, M.D., B.S., F.R.C.S. Pp. xlvi, 384. London: Oxford Medical Publications. 1910. Price 25s.—We regard this work as one of the most important monographs on the subject that has yet appeared, giving as it does the completest information as to the history, causation, bacteriology, pathology, symptoms and treatment of this affection. There are several excellent points in the arrangement of the work worth noting. The index is most complete and thorough, the plates are numerous and clear, showing a high degree of artistic merit, while the arrangement of the bibliographical references is most careful, a complete list being given at the end of each chapter. No reference of importance is omitted, and the large number quoted makes the book invaluable as a work of reference. The author's style is easy and pleasant, and the print large and clear. Space will not permit of such a full consideration of the contents as is demanded by their importance, but we would commend specially for study the statistical tables given, and the author's remarks thereon. He produces good evidence to show that the seasonal incidence of puerperal infection is a fact the explanation of which is comparatively simple, but none the less of first-class importance, and that while air-borne infection is comparatively uncommon, yet climatic conditions have a most important influence on the cure incidence of the disease. He also rightly lays stress on the frequency of the occurrence of infection in cases of toxæmia of other than septic origin. This fact, well known as it is to obstetricians, cannot be too strongly emphasised. His account of the bacteriology is most illuminating, and his insistence on the frequency of mixed infections and the infrequency of pure ones a point that requires general emphasis. He does not consider that the work so far presented on serum treatment is sufficiently definite in its results to allow of this method being regarded as a specific means of dealing with the affection, but his presentation both of this method and the treatment by vaccines is most valuable and complete. He gives a lucid and full account of the different operative procedures required, and lays down one sound rule which may render the decision as to whether hysterectomy is called for or not less difficult than it usually is. His suggestion is that the puerperal uterus should be looked upon in the same way as the non-puerperal, and that just as we should advise and practise hysterectomy in the case of an infected fibroid, so we should adopt a similar course in the case of a limited local infection of the puerperal uterus. We can only say that any

one who wishes to consult a work of reference on the subject will find no clearer or fuller information on any point he may wish to ascertain.

Transactions of the American Association of Obstetricians and Gynæcologists. Vols. XIX and XX. New York. 1906–7.—These volumes contain many interesting records of cases and discussions. Vol. XIX contains an interesting case and discussion of Porro Cæsarean section during pregnancy in a patient in whom irrigation of the membranes with prolapse of cord, but without labour pains, occurred in a patient suffering from a fibroid in the lower segment of the uterus, filling the pelvis completely. Both mother and child recovered. Several similar cases were narrated in the following discussion, which contained several references to myomectomies performed during pregnancy. Papers on "Chronic Dyspepsia resulting from Pelvic and Abdominal Disease" (Herman Hayd), "The Diagnosis and Treatment of Accidental Hemorrhage" (Adam H. Wright), "Anterior Vaginal Celiotomy" (Saml. Bandler), "Some Observations and Experiences Respecting the Symptoms and Treatment of Cases of Atresia of the Vagina" (Augustus Clarke), will all repay perusal. Amongst other papers in Vol. XX, one by Raleigh R. Huggins on "The Toxæmias of Pregnancy" deserves notice The author, among other acute observations, notes the liability of sufferers from pregnancy toxæmias to the development of septic infection in the puerperium, and also relates cases which throw some light on the curious cases of jaundice met with at times as a complication of the puerperium. Hayd relates the removal, with success, of a lithopedion from a woman aged 67. James W. West also in a paper on "When shall we Perform Myomectomy and when Hysterectomy in Uterine Fibromyomata?" gives the pros and cons of the merits of the respective operations with great clearness, and also lays down distinctly the rules which should govern the treatment by operation generally. James E. Sadlier on "Consistency in Aseptic Surgical Technique" makes some rather pregnant remarks on technique and consistency which will be found valuable. Edward J. Ill in a paper on "The Conservative Medical Treatment of Salpingitis" presents a valuable corrective to those who consider operation the one and only treatment for inflammatory affection of the uterine appendages. He gives one or two valuable suggestions as to treatment, and also as to cases in which reinfection sometimes periodic in character takes place. The paper is one well worthy of study.

Gynæcological Therapeutics. By S. JERVOIS AARONS, M.D., M.R.C.P. Pp. xiv. 178. London: Baillière, Tindall & Cox. 1910. 5s. net.—This little volume is written for the use of the general practitioner as a handy reference book, containing definite and

concise directions for the treatment of those gynæcological affections which fall within his province, and as such should be warmly welcomed by him. No account of pathology or diagnosis is attempted, the work dealing simply with the question of treatment. The result is that this book contains within small compass a collection of prescriptions, with practical hints on their use and the necessary instructions to patients, which must be most useful to anyone embarking on general practice with only the amount of knowledge and experience in this department acquired in the course of the ordinary curriculum. Particularly good are the chapters on affections of the external genitals and on constipation. The former furnishes the practitioner with a wide choice of remedies to which he may turn in those perplexing cases of pruritus, eczema vulvæ, etc., which at times tax his ingenuity in therapeutics, while the latter contains most helpful advice on the rational treatment of cases of simple constipation. Some useful prescriptions will also be found on those pages dealing with dysmenorrhœa, but no mention is made of guaiacum in the treatment of this complaint, although some cases are very markedly relieved by this drug. The fact that this little book contains at any rate a summary of all the medical side of gynæcological treatment, serves to show clearly how essentially surgical this department of our art has become.

Notes on Preparations for the Sick.

Phosphocose—Glycero-phosphate Somatose.—THE BAYER CO., London.—The various preparations of somatose have been noticed by us from time to time. The list now includes the guaiacol compound of calcium guaiacol-sulphonate called guycose, iron somatose, irocose=liquid iron somatose, liquid somatose, milk somatose; and to these is now added phosphocose. It is a brown syrupy liquid of pleasant flavour, and consists of a pleasantly-flavoured solution of somatose, containing sodium glycero-phosphate. It contains no alcohol or other preservative, but is sent out sterilised: it should therefore be prescribed and dispensed in the original bottles only. In cases of nervous debility and neurasthenia generally the combination of a soluble meat albumen with glycero-phosphate is an ideal fluid, which should have a very wide range of therapeutic utility.

Horlick's Malted Milk. Lunch Tablets.—HORLICK'S MALTED MILK CO., Slough.—These tablets, five-eighths of an inch in

diameter, are packed in a flat box, a quarter of an inch in thickness, so forming a vest pocket lunch for very busy people. They are supplied in natural flavour, or with chocolate, and the case is refilled from time to time from the larger glass jar. They may be swallowed or dissolved slowly in the mouth as desired.

Luminal, Luminal Sodium, Afridol Soap.—THE BAYER CO., London.—The luminal tablets contain a grain and a half of this powerful hypnotic, which is chemically phenyl-ethyl-malonyl-urea, a white odourless powder of somewhat bitter taste. It differs from veronal in having a phenyl group in place of the ethyl group.

The luminal sodium is a soluble form, which may be used hypodermically, the ordinary dose being 2.3 c.c. of 20 per cent. solution. The solution should be prepared in cool boiled water, and will keep for about eight days.

These drugs should be given cautiously, the maximum dose should not exceed twelve grains. In mental cases, alcoholics, drug habitués, etc., they have given excellent results, and may largely replace scopolamine.

Afridol soap is stated to be strongly germicidal, and thus useful for disinfecting the hands, instruments, and skin generally. It is not irritating, and does not injure instruments.

Euthymol—Tooth Paste, Cold Cream, Shaving Cream, Talcum Powder, Tooth Powder, Bath Crystals.—PARKE, DAVIS AND CO., London.—All these preparations have as their essential basis a proportion of this well-known antiseptic called euthymol. In the tooth paste it is equivalent in germicidal power to a 14 per cent. solution of carbolic acid, and although the mouth can never be absolutely sterilised, yet much can be done by chemical and mechanical methods combined to counteract the effects of pyorrhœa and other septic conditions of the mouth and throat. The euthymol paste is non-irritating and non-poisonous, but is pleasant to the taste, and thoroughly efficient as an oral germicide.

Chrismol.—ALLEN & HANBURY, London.—A liquid paraffin of the highest purity, intended for internal use. As such it may be of some mechanical advantage to the alimentary canal, and indirectly may improve nutrition; but we have always held that petroleum internally must be of little use, inasmuch as it passes through the alimentary canal unchanged. Patients say they derive benefit from its use, and that they gain in weight

thereby: but much evidence is required to verify these statements. If paraffin be the thing, surely this preparation must be the best form of it.

Novatophan.—A. & M. ZIMMERMANN, 3 Lloyd's Avenue, London, E.C.—In Dec., 1912, we noted the chief points relating to a phenylchinoline carbonic acid compound named atophan. It has been found to have a powerful influence on uric acid metabolism, increasing the uric acid elimination remarkably.

The newer form is the ethyl ester of methylated atophan, which is the equivalent in clinical effect, but without the bitter taste. It is not soluble, and therefore the tablet form is advised, six to ten being the usual daily dose (45 to 75 grains).

Pond's Tampons.—Local Medication for Women.—J. M. RICHARDS, London.—These vaginal tampons are easily applied without speculum. Moistened in warm water for about ten seconds and then inserted, being held in place for a few moments, it will be found that the gelatine shell soon dissolves, the expanding wool forcing the medicated cone against the cervix, and dilating the vaginal walls. They are made in three sizes, and contain various medicated compounds such as ichthyol compound with boro-glyceride, protargol, tannin, and opium, belladonna, and hyoscyamin with a boro-glyceride base. The medicated tips are completely dissolved in about 24 hours, when the tampon should be changed.

Ems Mineral Spring Salt Pastilles.—HERTZ & CO., 9 Mincing Lane, London.—This natural salt is obtained by evaporation of the water of the Ems spring at its source, under control of the Prussian State.

Lactona.—H. E. MATTHEWS & CO., Clifton.—This is a tonic milk powder, prepared by a local firm, and differing from many similar preparations. In the first place our analysis showed it to contain over 20 per cent. of fat, and this alone places it in a class by itself. It is also extremely palatable, and is especially pleasant when milk is used as the vehicle for its administration. Unlike the majority of tonic foods the price is low, a half pound tin costing only 2s. The addition of 5 per cent. sodium glycerophosphate largely increases the nutritive value of the other constituents.

The Library of the Bristol Medico-Chirurgical Society.

The following donations have been received since the publication of the List in March.

May 31st, 1913.

F. R. Cross (1) .. .. 3 volumes.
L. M. Griffiths (2) .. .. 12 ,,
E. W. Hey Groves (3) .. .. 1 volume.
A. H. Hooker (4) .. .. 1 ,,
R. Shingleton Smith, M.D. (5) .. .. .. 14 volumes.

Unbound periodicals have been presented by Mr. F. R. Cross and Mr. J. C. Heaven. Reprints have been received from Dr. James Swain.

EIGHTY-EIGHTH LIST OF BOOKS.

The titles of books mentioned in previous lists are not repeated.

The figures in brackets refer to the figures after the names of the donors, and show by whom the volumes were presented. The books to which no such figures are attached have either been bought from the Library Fund or received through the *Journal*.

Abel, R.... .. .. *Laboratory Handbook of Bacteriology* (Trans. from fifteenth German edition by M. H. Gordon) .. .. 2nd Ed. [1912]
"Æsculapius Scalpel." *Dying Scientifically* .. .. (2) 1888
Alderson, W. E. .. *Dental Anæsthetics* .. .. 2nd Ed. 1913
Barker, W. R. .. *The Bristol Museum and Art Gallery* 1906
Batten and H. Thursfield, A. E. Garrod, F. E. [Eds.]. *Diseases of Children* 1913
Birmingham, Handbook of (B.M.A.) 1911
Bland-Sutton, Sir J. *Fibroids of the Uterus* .. .. [1913]
Brody, G. St. .. *The Flora of Weston* .. .. (2) 1856
Browne, Sir T. .. *Religio Medici* (Ed. by W. P. Smith) .. (2) 1874
Burghard, W. W. Cheyne and F. F. *Manual of Surgical Treatment.* New Ed. (Ed. by T. P. Legg and A. Edmunds).. .. .. Vol. IV 1913
Burnet, E. .. .. *The Campaign against Microbes* (Trans. by E. E. Austen) .. .. (5) 1909
Cheyne and F. F. Burghard, W. W. *Manual of Surgical Treatment.* New Ed. (Ed. by T. P. Legg and A. Edmunds).. .. .. Vol. IV 1913
Chundra, J. L. .. *Laws of Sexual Philosophy* 1913
Cotar, C. .. .. *The Mineral Waters of Vichy* 1913
Cornaro, L. .. .. *Sure Methods of attaining a Long and Healthful Life* (Trans. by W. Jones) .. .. (2) 1768

Crooke, G. F. .. *Medical Pathology of Tuberculosis* .. (5) 1891
Culpeper, N. .. .. *The English Physician* (enlarged) .. (2) 1801
Dührssen, A. .. *Geburtschilfliches Vademekum* .. 10th Ed. 1913
Faust, B. C. .. *Catechism of Health* .. .. .. (2) [1832]
Fernie, W. T. .. *Our Outsiders: and what they betoken* 1913
Flint, A. .. .. *Medicine of the Future* .. .. (5) 1886
Freud, S. .. .. *The Interpretation of Dreams* (Trans. by A. A. Brill) 1913
Gabbett, P. C. .. *Manual for Women's Voluntary Aid Detachments* .. .. 2nd Ed. [N.D.]
Garrod, F. E. Batten and H. Thursfield, A. E. [Eds.] *Diseases of Children* 1913
Goodhart, Sir J. F. *The Passing of Morbid Anatomy* .. .. (5) 1912
Hanna, W. .. .. *Studies in Small-Pox and Vaccination* 1913
Hart, A. H. .. .. *How to Cut the Drug Bill* .. .. 3rd Ed. 1913
Hewer, Mrs. J. L. *Our Baby* .. .. 14th Ed. 1913
Hooker, A. H. .. *Chloride of Lime in Sanitation* .. .. (4) 1913
James, A. .. .. *Pulmonary Phthisis* .. .. (5) 1888
Jex-Blake, A. J. .. *Death by Electric Currents and by Lightning* 1913
Johnson, J. .. .. *Excursions to the principal Mineral Waters of England* .. .. (2) 1843
Jones, H. L. .. *Medical Electricity* .. .. 6th Ed. 1913
Julliard, C. .. .. *Les Bandages* .. .. 2nd Ed. 1913
Langmead, F. .. *Forms of Mental Defect* 1913
Milne, R. .. .. *Home Treatment and Prevention of Scarlet Fever* .. .. (5) 1910
Molière, J. B. P. de *Le Médicin malgré lui* (Notes by G. E. Fasnacht) .. .. (2) 1895
Morland, C. Riviere and E. *Tuberculin Treatment* .. 2nd Ed. 1913
Murphy, J. K. [Ed.] *The Practitioner's Encyclopædia of Medicine and Surgery* .. .. 2nd Ed. 1913
Musgrove, C. D. .. *Nervous Breakdowns and How to Avoid Them* 1913
Parker, T. J. .. *Biographical Sketch of W. Kitchen Parker* (5) 1893
Pembrey and J. Ritchie, M. S. [Eds.] *General Pathology* 1913
Ritchie, M. S. Pembrey and J. [Eds.] *General Pathology* 1913
Riviere and E. Morland, C. *Tuberculin Treatment* .. 2nd Ed. 1913
Saundby, R. .. .. *Old Age* 1913
Sawyer, Sir J. .. *Insomnia* .. .. 2nd Ed. 1912
Sewill, H. .. .. *Dental Caries and Prevention* (5) 2nd Ed. 1888
Simon, A. L. [Ed.] *In Vino Veritas* .. .. (5) 1913
Thorne, L. T. .. *The "Nauheim" Treatment of Diseases of the Heart* .. .. 4th Ed. 1913
Thursfield, A. E. Garrod, F. E. Batten and H. [Eds.] *Diseases of Children* 1913
Tunstall, F. J. Warwick and A. C. *First Aid* .. .. 8th Ed. 1913
Walsh, D. .. .. *The Röntgen Rays in Medical Work* .. (5) 1897

Warwick and A. C. Tunstall, F. J. *First Aid* .. .. 8th Ed. 1913
Williams, C. T. .. *Old and New Views on the Treatment of Consumption* .. .. (5) 1911

TRANSACTIONS, REPORTS, JOURNALS, &c.

American Association of Genito-Urinary Surgeons, Transactions of the.. .. .. Vol. VII 1912
American Journal of Ophthalmology, The .. .. Vol. XXIX 1912
American Journal of Surgery .. .. Vol. XXVI 1912
American Laryngological, Rhinological and Otological Society, Transactions of the 1912
American Medicine .. .. Vol. XVIII 1912
American Pediatric Society, Transactions of the .. Vol. XXIV 1912
Annales de Dermatologie et de Syphiligraphie .. Tom. III 1912
Annales d'Oculistique .. .. (1) Tom. CXIII, CXIV 1895
Archives of Pediatrics .. .. Vol. XXIX 1912
Boston Medical and Surgical Journal, The .. Vol. CLXVII 1912
British Journal of Dermatology, The .. .. .. Vol. XXIV 1912
British Journal of Tuberculosis, The .. .. .. Vols. V, VI 1911
Bulletin de l'Académie de Médecine .. Tom. LXVII, LXVIII 1912
Bulletin de la Société française de Dermatologie 1912
Canadian Medical Association Journal, The .. .. Vol. II 1912
Clifton College Register .. .. (2) 1880, 1887, 1890
Clinical Journal, The .. .. Vol. XL 1912
College of Physicians of Philadelphia, Transactions of the Vol. XXXIV 1912
Deutsche Zeitschrift für Chirurgie .. .. Bd. CXIX 1912
Dublin Journal of Medical Science, The .. .. (2) Vol. LXX 1880
Écho médical du Nord, L' 1912
English Catalogue of Books, The 1912
Gazette des Hopitaux 1912
Gazette des Hopitaux de Toulouse 1911
Hospitals—
Johns Hopkins Hospital Reports, The .. .. Vol. XII 1904
St. Bartholomew's Hospital Reports .. .. Vol. XLVIII 1913
Jahreskurse für Ärztliche Fortbildung .. .. (2) 1913
Johns Hopkins Hospital Bulletin, The .. .. Vol. XXIII 1912
Journal of Experimental Medicine, The .. .. .. Vol. XVI 1912
Journal of Laryngology, Rhinology and Otology, The Vol. XXVII 1912
Journal of Nervous and Mental Disease, The Vol. XXXIX 1912
Journal of Obstetrics and Gynæcology of the British Empire, The Vol. XXII 1912
Journal of the American Medical Association, The Vol. LIX 1912
Journal of the Royal Sanitary Institute .. .. Vol. XXXIII 1912
Journal of Tropical Medicine, The .. .. Vol. XV 1912
Library Association Record, The .. .. Vol. XIV 1912
Medical Annual, The 1913

Medical Record	Vols. LXXX, LXXXII	1911–12
Münchener medizinische Wochenschrift	Bd. II für	1912
National Association for the Prevention of Consumption, Transactions of the	(5)	1911
Post-Graduate, The	Vol. XXVII	1912
Practice of Medicine and Surgery by Unqualified Persons in the United Kingdom, Report as to the		1910
Public Health	Vol. XXV	1911–12
Revue de Chirurgie	Tom. XLVI	1912
Revue de Thérapeutique médico-chirurgicale		1912
Revue générale d'Ophtalmologie	Tom. XXXI	1912
Therapeutic Gazette, The	Vol. XXXVI	1912

MEETINGS OF SOCIETIES.

Bristol Medico-Chirurgical Society.

March 14th, 1913.

Dr. WALTER C. SWAYNE, President, in the Chair.

Dr. P. WATSON-WILLIAMS showed a case, one year after removal, of an **Extensive epithelioma of the larynx** without recurrence, but with some stridor from cicatricial contraction.

Mr. HUBERT CHITTY showed an infant, aged six weeks, with on the left side **Congenital absence of the ulna and two digits with their metacarpals,** and on the right side absence of **one digit and its metacarpal.**

Dr. CAREY COOMBS, in a paper on **Certain adventitious pericardiac sounds,** pointed out the frequent occurrence in cases of cardiac valvular lesions of pulmonary crepitations over the third left interspace in front, particularly in cases of mitral stenosis. He said that these were probably due to collapse of the lung margin, owing to pressure of the enlarged conus arteriosus, together with hyperæmia, and were of interest in that they not uncommonly led to a mistaken diagnosis of pulmonary tuberculosis. The stationary condition of the signs, together with the absence of tubercle bacilli in the sputum, were sufficient to negative the presence of tuberculosis.—Dr. J. MICHELL CLARKE remarked that the limitation of these crepitations to the first deep inspiration during auscultation pointed to their being due to a condition of atelectasis.—Dr. J. R. CHARLES, in an examination of some thousands of post-mortem records, had found that the coexistence of pulmonary tuberculosis and mitral stenosis was a rare occurrence.

Dr. CHARLES read a paper on the **Etiology of migraine,** in which he propounded the theory that the symptoms of migraine were due to a transient swelling of the pituitary gland. Such swelling was known during pregnancy, and from the anatomical position of the gland it was conceivable that if swollen it might produce effects on the optic nerves and those in the walls of the cavernous sinus. The vasomotor effects might be explained by an increased secretion from the posterior lobe.—Mr. H. ELWIN HARRIS did not think that the theory could explain in his own case the occurrence of hemianopsia half an hour before the headache.—Dr. COOMBS did not see how the association of migraine and epilepsy could be explained by this theory.

Dr. WATSON-WILLIAMS read notes and showed a specimen from a case of **Cerebro-spinal rhinorrhœa,** in which there was an opening found in the dura and bone of the anterior fossa post-mortem, death occurring from meningitis from a complicating frontal sinusitis. During life the cerebro-spinal fluid had caused a fluctuating swelling at the inner orbital margin, thus simulating an ethmoidal mucocele.

April 9th, 1913.

Dr. WALTER C. SWAYNE, President, in the Chair.

Mr. E. H. E. STACK, in a demonstration on some varieties of **Arm splints,** advocated the use of a malleable metal anterior angular splint for the treatment of fractures of the forearm. For all injuries about the elbow-joint he had found the position of acute flexion to give the best results, and demonstrated a simple method for putting up an arm in that position. He advocated the early use of active rather than passive movements, the patient being better able to judge the amount of movement possible without risk of damage.

Mr. CHARLES CORFIELD, read a paper entitled **"What is an Accident?"** (*vide* p. 148).

Mr. W. STUART V. STOCK related two cases in which death occurred two or three days after an operation from **Heart failure** in patients the subject of fatty heart, although they had shown no bad signs during a prolonged administration of an anæsthetic.

Mr. A. RENDLE SHORT, in a paper on the **Nature of surgical shock,** pointed out that the two great objections to the generally-accepted theory of Crile and Mummery—that it was due to vaso-motor centre fatigue—were that the peripheral vessels were found to be constricted and not dilated, and also that the centre could be shown experimentally not to be exhausted. He had examined the venous blood of a number of patients the subject of shock, but had been unable to confirm the theory of Yandel

Henderson that shock was due to a deficiency of CO_2 in the blood. His own view was that the symptoms were due to a loss of fluid from the blood which apparently passed out into the tissues and was possibly squeezed out by vasomotor reflexes.

May 14th, 1913.

Dr. WALTER C. SWAYNE, President, in the Chair.

Dr. CAREY COOMBS showed a case of **Congenital Cardiac lesion.** The patient, a boy of four, had been first noticed to get blue at times when six months old. He had clubbed fingers and cyanosis, and was undergrown. The cyanosis was paroxysmal, and induced by exertion and with colds. The heart was enlarged to right and to left, and had a loud rasping bruit. The speaker gave reasons for assuming the lesions to be congenital pulmonary stenosis, coupled with patency of the interventricular septum. He considered the prognosis to be bad.—Dr. EVERY CLAYTON thought the boy had adenoids, and that oxygenation would be improved if they could be safely removed.

Dr. E. C. WILLIAMS showed three cases: (1) **Ataxia** in child of three. The child could only stand if supported. The gait on attempting to walk was characteristically ataxic. The knee jerks were exaggerated. The plantar reflexes flexor. No optic atrophy or neuritis was present. The condition seemed stationary, not advancing as in a Friedrich's ataxy. (2) A case of the **Juvenile type of muscular atrophy.** The boy was eight years old. His health had been good until two years of age. He began then to fall easily, especially if tired. During the last six weeks he had shewn some hypertrophy of the calves. He had not considered the case to be one of pseudohypertrophic paralysis. (3) A case of hepatic enlargement with ascites, and a purpuric eruption. Illness began with abdominal pain and hæmatemesis. Large quantities of fluid had been withdrawn from the abdomen repeatedly. There appeared to be portal obstruction.—Dr. PARKER thought the duration of the trouble was greater than that in cases of Henoch's purpura.—Dr. MICHELL CLARKE suggested a diagnosis of congenital syphilis of the liver.—Dr. EVERY CLAYTON said he had recently seen a case of purpura, the purpuric spots coming out in successive crops, which had lasted six months. Dr. STACK thought granular kidney might be the cause.—Dr. C. WILLIAMS replied that he had purposely named this case the juvenile type of muscular atrophy, as the case was of long standing (two years). The muscular weakness was rapidly advancing, and in a typical case of pseudohypertrophic paralysis there would be well-marked enlargement of the buttocks. There is no essential difference between the two diseases; they are probably only

modifications of the same disease, so there can be no possible reason in calling the case pseudohypertrophic, when the only pseudohypertrophy which exists is that found in the juvenile type of muscular atrophy.

Dr. D. A. ALEXANDER showed a case of **Brachial hypertrophy.** The youth had massive upper limbs and slight enlargement of the right leg, but a small head. The suggestion was made that the boy's condition illustrated reversion to an ancestral, less developed type of human being.—Dr. CLARKE thought there might be some pituitary gland change causing the hypertrophy.

J. LACY FIRTH.
A. J. WRIGHT, *Hon. Sec.*

Obituary.

DR. STANLEY JENKINS.

IT is with very deep regret that news has been received of the sad death of Dr. H. Stanley Jenkins, one of the most distinguished students of the Bristol Medical School. He was the son of Mr. F. A. Jenkins and the great-nephew of the late Dr. Robert Fletcher, of the Surgeon-General's Library, Washington. After working at the Bristol General Hospital, Dr. Jenkins took the Conjoint Qualification in 1898, proceeding in 1903 to the M.D. London and the F.R.C.S. Since 1904 he had devoted himself to the life of a medical missionary in China, where in April of this year he succumbed to an attack of typhus fever. From Dr. Thomas Scollay, a colleague of Dr. Jenkins's at the English Baptist Mission, Sianfu, Shensi, North China, we have received the following short account of Dr. Jenkins's work and last illness, which we are glad to be able to publish:—

"On the 6th of April Dr. H. Stanley Jenkins, of the Baptist Missionary Society, passed away in his thirty-ninth year at the Mission Hospital, Sianfu, Shensi, China. He had just shortly returned to the hospital which he had been in charge of for nearly nine years.

"He first arrived in the year 1904, when China was just settling down after the Boxer rising, and the natives were beginning to realise that the Foreign Missionary only desired to help them. The Chinese among whom he worked will miss him much, for they recognised his skill in treating them when they needed his help, and at every time found in him a true friend whom they could consult on matters not medical. The

Society to which he belonged will miss him much, for while men of his ability are not too numerous, there are few who are so willing to give their years and talents to practising in the heart of China.

"In 1904 there were few railways in China, and the inland journey to Sianfu then took a good month. Immediately on his arrival Dr. Jenkins found his hands full of work, but with a willing heart he took up his task. The difficult language, the despair of so many, proved easy to him, and he soon won the hearts of the people. There is nothing that pleases a Chinese so well as finding someone who is willing to listen to his story. Dr. Jenkins proved a patient listener, and it did one good on out-patient days to see him never showing a trace of weariness as a long story was poured into his ears, most of it irrelevant to the case in question.

"Medical work in Sianfu is very different to what one is accustomed to in England. To begin with, the cases one sees are different. Of course, the Chinese have the same diseases as the English, with some few additions, but those same diseases are seen very often in more advanced stages. Wounds are seen full of maggots, and malignant disease rarely seen until long past the operable stage. The reason for this is that the foreign doctor is looked upon as the last hope. Very often every means of native help is first sought, and only the most serious cases fall into the missionaries' hands. A man will come with a plaster covering his shoulder. This is the native treatment for dislocated shoulder-joint, and has been tried for perhaps six months before the Mission doctor sees it. Besides the plaster the native doctor has one other method of treating such surgical cases. They insert needles to let out the evil humours. Thus it was in surgery that Dr. Jenkins first found scope for his energies.

"The native doctor is not without some knowledge of drugs and their uses. His medicine chest contains many common vegetable aperients, digitalis and many others in the B.P., but all in very crude form. An infusion of the herb in a pint of water would generally be the dose. This proved a great difficulty when using foreign concentrated drugs. The handy little tablet proved rather a drawback.

"There were other and greater difficulties, however. The pioneer doctor finds that he must be surgeon, house-surgeon, nurse and ward servant until he has trained his own assistants. He must also dispense his own medicines, but fortunately the Chinese take well to dispensing. Antiseptic methods, however, are by no means easy for them to learn, and so naturally cases go septic much more readily than at home. Dr. Jenkins, therefore, found his operations limited until he had trained the Chinese to be efficient. The Chinese, Dr. Jenkins found at first,

were rather averse to operations of a major type. They had a very great dislike to the loss of limbs, and only consented when at last they discovered that lives were saved by it. It is also practically a new thing to get permission to open an abdomen. The foreign doctor found that some of his practice came to him because he had chloroform, or 'dreaming medicine' as they call it.

"This, then, was the pioneering work that Dr. Jenkins was engaged in before he went home for his first furlough in 1911. During his furlough he was as enthusiastic as ever in the interests of his beloved hospital. He made arrangements for installing an X-ray apparatus, and it is sad to think that the apparatus was well on its way out to him when he became ill. Other improvements were also thought over and arranged, the chief one being the building of a much-needed new hospital.

"When at home for a short time the news of the revolution came, and Dr. Jenkins volunteered to return to relieve the little band who were holding the fort. News came, however, that most of the missionaries were leaving for the coast, and so he remained at home for full furlough.

"On his return to Sianfu Dr. Jenkins found the work carried on by his colleague, Dr. Robertson, and immediately took up his share. In less than two months Dr. Robertson became ill with typhus fever. In addition to the whole hospital work that fell on his shoulders, Dr. Jenkins took his share in nursing the patient. Dr. Robertson had got the disease in the out-patient department, and subsequent events showed that Dr. Jenkins had also got the disease from the same source. At the end of four days Dr. Jenkins became ill, but he continued to work without letting anyone know. On the Friday, with a temperature of 103 deg., he saw about sixty out-patients. That night he went to bed with his Chinese servant sleeping in his room as the only attendant. Next day he was unable to get up. It was good to see how his Christian faith made him at once reconciled to God's will. He knew that his heart bore traces of former rheumatism; but if it was the call to other service he was ready to go. Only his ties to his wife and two little children made him hesitate for a moment, but he soon gained strength to leave them to the keeping of that same Jesus who was calling him.

"Two weeks passed and the fever left him. We thought he was going to be left to us here, but the strain upon his heart had been too much, and a week later he passed away. His wife and some of his fellow-missionaries were at his bedside. God was good in allowing Mrs. Jenkins, after a fortnight's journey, to spend the last three days with her husband.

"He was laid to rest in the Baptist Missionary Churchyard by his fellow-missionaries and many of the Chinese to whom he

had brought the Gospel, and laid down his life in giving it. What a sad blank he has left in their lives and ours, whom he has left to perhaps reap some of what he has sown."

Local Medical Notes.

University of Bristol.—Students of the University have recently passed the following examinations :—

University of London.—*Second Examination for Medical Degrees : Part I*, C. C. Chesterman. *Part II*, A. W. Adams ; (excluding pharmacology), F. K. Hayman.

M.B., Ch.B. Bristol.—*Intermediate Examination :* C. N. Vaisey, Hilda K. Ewins, W. McKenzie, G. R. Wookey.

Conjoint Board of London.—*Chemistry and Physics :* F. L. Fox, A. E. Young. *Biology :* W. A. Jackman. *Practical Pharmacy :* W. E. Neale. *Anatomy and Physiology :* H. R. W. Husbands, R. W. Little, D. Munro-Smith. *Medicine, Surgery and Midwifery :* W. Salisbury.* *Medicine :* H. W. Cooke, Miss Barnes. *Surgery :* Miss Barnes. *Midwifery :* A. G. Morris, R. B. Johnson, H. N. Morgan.

L.D.S. Eng.—*Mechanical Dentistry :* Marjorie J. White. E. W. Joyner. *Dental Metallurgy :* E. W. Joyner, W. H. Greet. *Final Examination (Part I) :* E. W. Thomas, J. D. Melhuish, *Part II :* K. Batten,* J. D. Melhuish.*

APPOINTMENTS.

Bristol General Hospital.—C. A. Morton, F.R.C.S., has been re-appointed Surgeon. E. W. Hey Groves, M.D., M.S. Lond., F.R.C.S., has been appointed Surgeon. A. J. Wright, M.B., B.S. Lond., F.R.C.S., Surgeon to the Nose and Throat Department ; and Clifford A. Moore, M.S. Lond., F.R.C.S., Assistant Surgeon ; J. Freeman, M.D., has been re-appointed Anæsthetist.

G. Hely-Hutchinson Almond, B.M., B.Ch. Oxon., has been appointed Honorary Pathologist to the Royal Mineral Water Hospital, Bath.

C. A. Joll, F.R.C.S., has been appointed Senior Resident Medical Officer, Royal Free Hospital.

A. Fells, M.B., C.M. Edin., has been appointed Honorary Surgeon to the Bristol Eye Dispensary.

* Qualified.